The Interpretation of the

UNIPOLAR ELECTROCARDIOGRAM



ILLUSTRATED



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PREFACE

This work is the outgrowth of previous manuals prepared to supplement the *Graduate Course* in Electrocardiography given at Wayne University College of Medicine for many years The objective is an organized presentation of the interpretation of the unipolar electrocardiogram The only basic physics included is that considered essential to the interpretation of the tracing controversial theories that do not contribute to clinical analysis have been omitted The vast literature has been utilized freely as a background but individual references have not been cited in order to conserve space

The analysis of the arrhythmias is made from leads that clearly display auricular as well as ventricular deflections the interpretation of the QRS-T complex is based upon the findings in multiple precordial and back leads supplemented when necessary by esophageal and unipolar limb leads The registration of the normal and abnormal QRS-T complex is based upon concepts developed by Frank N Wilson and associates and shown to be consistent with experimental and post mortem findings These concepts have been presented in detail and suitably illustrated with diagrams as an introduction to clinical analysis Efforts have been concentrated on a systematic lucid description of electrocardiographic findings in an attempt to make reproduction of tracings unnecessary

The descriptions are drawn in part from the literature in part from observations derived from correlation of electrocardiographic and post mortem findings carried out at Detroit Receiving Hospital during the past fifteen years The author is particularly indebted to Dr Tomiharu Hiratzka Associate Professor of Pathology who has devoted especial attention to the autopsy findings Invaluable assistance has been given by numerous associates in the Department of Medicine especially Dr Charles H Sears formerly Assistant Professor of Medicine and Dr Muir Clapper Professor of Medicine The diagrams were made by Miss Evelyn Erickson and Mrs Geraldine Fockler This work has been supported in part by grants from the Michigan Heart Association

Electrocardiographic interpretation in this manual has been approached as an entity in order to facilitate description and evaluation of its contribution to Cardiology In the practice of medicine the electrocardiogram should be employed as an adjunct in the final diagnosis and never as a substitute for clinical findings it should always be interpreted in the light of all clinical data and never in the absence of other information Under these circumstances electrocardiographic interpretation achieves its proper role in clinical medicine

G B Myers

TABLE OF CONTENTS

The Interpretation of the

UNIPOLAR ELECTROCARDIOGRAM

A. INTRODUCTION THE RELATION OF THE ELECTROCARDIOGRAPHIC DEFLECTIONS TO MECHANICAL AND ELECTRICAL EVENTS IN THE CARDIAC CYCLE

The electrocardiogram is a record of potential variations originating in the heart and registered from arbitrary points on the body surface or within the esophagus The relationship of electrical to mechanical events in the cardiac cycle is illustrated by Figs 1A and 1B The former represents a simultaneous recording of standard Lead I of the electrocardiogram heart sounds and jugular pulse whereas the latter is similar except that carotid pulse is recorded in place of jugular pulse

<u>T P segment</u> At heart rates below 100 there is an appreciable interval following the second heart sound during which the entire heart is normally at rest both electrically and mechanically Throughout this interval the string remains stationary as evidenced by the fact that its photographic image pursues a horizontal course the isoelectric line (T-P segment) The smooth horizontal level indicates maintenance of a constant potential During the inscription of the isoelectric line under normal circumstances a zero potential prevails throughout the heart and galvanometric circuit * The ventricles are in diastole during the registration of the isoelectric line as evidenced by a progressive fall in carotid pressure (Fig 1B) The jugular pressure rises early in the period as a result of auricular filling (V wave) then falls with the opening of the auriculoventricular valves (Fig 1A)

<u>P wave</u> The isoelectric line is terminated by the advent of the P wave recorded as a gradually sloping rounded upright deflection in leads facing the ventricular surface Coincident with the terminal portion of the P wave there is a sharp rise in jugular pressure due to auricular contraction (Fig 1A) This fixed relationship indicates that the P wave represents the intra-auricular spread of the impulse which initiates auricular contraction Following the end of the P wave there is a brief interval corresponding to the passage of the impulse through the auriculoventricular node During this interval the string shadow usually drops slightly below the isoelectric line as a result of the beginning of the auricular T wave which is produced by repolarization of the auricles and is analogous to repolarization of the ventricles to be discussed in more detail

<u>QRS complex</u> The next event produces the most prominent feature of the electrocardiogram and is manifested by one or more sharp spikes referred to collectively as the QRS complex Only two of the three classical phases of the QRS are represented in the illustration The portion of the deflection which is above the isoelectric line is designated as the R wave and is composed

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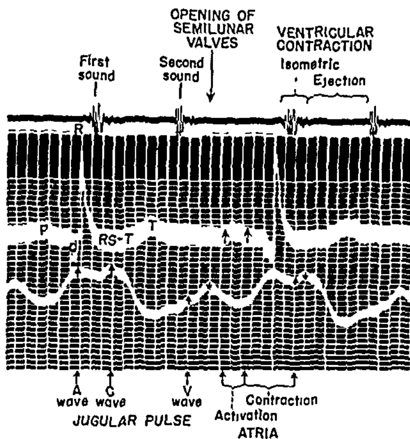


Figure 1A

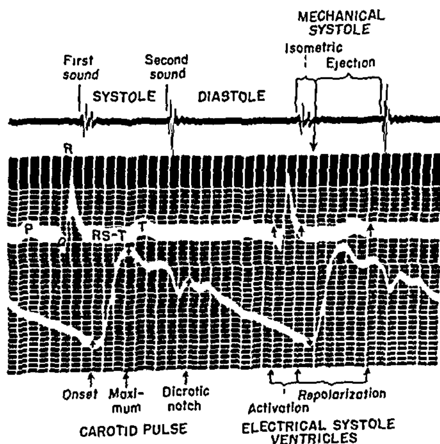


Figure 1B

of two limbs an ascending and descending The latter is precipitous in precordial and esophageal leads and is known as the intrinsicoid deflection By definition an initial downstroke which precedes the R wave is designated as a Q wave a secondary downstroke which follows the intrinsicoid deflection below the isoelectric line is known as an S wave This tracing displays a diphasic qR deflection and shows slurring at the base of the descending limb of the R wave in place of the S deflection When only one phase is present it is referred to as an R wave if the deflection is upright and as a QS complex if the deflection is entirely below the isoelectric line When the QRS is multiphasic and includes two upright deflections the second upstroke is referred to as an R' deflection Another downstroke extending below the isoelectric line after the R' wave is designated as an S' deflection

The Q wave and the ascending limb of the R are completed prior to the onset of mechanical systole as is evident from a study of Fig 1B The first heart sound marking the onset of ventricular contraction begins during the registration of the descending limb of the R wave whereas the abrupt rise in carotid pressure marking the ejection phase begins near the termination of the QRS complex These relationships indicate that the QRS complex represents the intraventricular spread of the activating impulse which initiates ventricular contraction

S T complex At the end of the QRS complex customarily referred to as the S-T junction the string shadow normally returns to the isoelectric line for a brief but variable period then produces a gradually sloping upright deflection of smooth contour known as the T wave The electrical phenomena which give rise to the T wave occur during ventricular systole as shown by the fact that the T wave is completed just before the advent of the second heart sound (Fig 1B) If the countless cells comprising the ventricles were likened to minute storage batteries connected in series and capable of rapid discharge and spontaneous recharge the QRS complex would represent the fluctuations in potential created by discharge or depolarization the T wave the potential changes associated with recharge or repolarization

U wave Following the termination of the T wave and the onset of the second heart sound an additional small wave known as the U wave may be present The origin of the U wave is unknown but the prevailing concept is that it represents an after-potential in the ventricle analogous to that which has been demonstrated in the nerve after the conduction of an impulse

B. GENERAL PROCEDURE FOR THE INTERPRETATION OF MULTIPLE PRECORDIAL AND UNIPOLAR LIMB LEADS

√I PRELIMINARY SURVEY to determine if the tracing is technically satisfactory and if a sufficient number of leads has been obtained

A EVALUATION OF TECHNICAL QUALITIES OF EACH LEAD

1 The standardization record produced by the sudden introduction of a potential difference of exactly 1 0 millivolt (mv) into the circuit is characterized by an abrupt vertical displacement of the baseline at the moment of application and an abrupt return to the original level at the moment of release Standardization should be recorded between the completion of the T wave and onset of the P wave of the next cycle while the electrocardiographic tracing is pursuing a horizontal course otherwise allowance must be made for the distortion caused by the superimposed potential changes of cardiac origin Either the vertical jump made by the introduction of the known potential difference or that registered by its removal may be selected for the following measurements depending upon which is recorded during the T-P segment or during the more stable portion of the cardiac cycle

a Amplitude In a correctly standardized tracing the vertical jump should measure 1 0 cm in which event each millimeter of the electrocardiographic deflections will represent a potential difference of 1 mv If the standardization record measures less than or slightly more than 10 mm the true voltage of the electrocardiographic deflections may be calculated by multiplying their measured amplitude by a fraction in which 10 is the numerator and the height of the standardization jump in millimeters is the denominator If the amplitude of the standardization record is 15 mm or more the tracing should be rejected because of the likelihood of artefacts from overshooting

b Time elapsing between onset and completion of the vertical jump should measure less than 02 sec When the standardization response requires more than 02 sec the registration of the QRS will be erroneously prolonged and distorted necessitating rejection of the tracing When the standardization record is made during the T-P interval the thin vertical line should terminate at an abrupt right angle with the thick horizontal line when made during the registration of the P or T wave the latter should continue undistorted from its point of interruption

2 Freedom from artefacts that interfere with interpretation Each lead should contain a minimum of 2 or 3 consecutive artefact-free cycles in which the T-P segment maintains a horizontal plane so that the position of the S-T junction in reference to the T-P segment may be accurately determined The commoner artefacts comprise

a Erroneous standardization with consequent QRS-T distortion is recognized from the standardization record Isolated abnormalities of amplitude that are within bounds

as described above may be corrected arithmetically An interval of 02 sec or more between onset and completion of the standardization jump is a manifestation of high skin resistance usually from poor electrical contact between electrode and skin and necessitates repetition of the electrocardiogram after proper application of electrode jelly Overshooting the combination of prolongation of the standardization jump exaggeration of its amplitude and the production of additional deflections demands rejection of the tracing

b Wandering baseline going up or down hill is usually *traceable to dirty* electrodes *which* act as a small battery and give rise to a minute current that flows through the electrocardiograph

c Irregular bizarre deflections may result from dirty electrodes loose *connections or* defects within the electrocardiograph

d Mixed up lead wires reverse the polarity and result in reciprocal deflections

e Alternating current interference is recognized by regularly spaced spikes *occurring* at 60 cycles per second when derived from the building *circuit, it is* eliminated by adequate ground connections and if necessary by removal of nearby electric motors

f Muscle tremor is manifested by irregular jagged oscillations associated with fine to coarse muscular contractions

B AVAILABILITY OF A SUFFICIENT NUMBER OF LEADS FOR A FULL EXPLORATION OF THE CARDIAC SURFACE Since a semidirect unipolar lead from the chest wall reflects principally the potential variations of the underlying epicardial surface multiple leads are obligatory to properly explore *each ventricle* The customary 6 precordial leads (V_1-V_6 incl) fail to cover the outflow tract of the right ventricle the base *of the* anterior and lateral walls and the entire posterior wall of the left ventricle and the diaphragmatic surface *of* the heart even when the heart is *normal in* size and position The following leads will provide adequate exploration in the *presence of most* abnormalities in size and position and are recommended as routine *Leads* V_{3R}-V_8 *inclusive* Leads HV_1-HV_8 inclusive taken at the intersections of vertical lines through the customary electrode positions with a horizontal line at the level *of the* sternal margin *of the third interspace* and Leads aV_R, aV_L and aV_F Unless the heart is considerably enlarged or markedly rotated, the potential variations *of the* tricuspid orifice are usually reflected in V_{3R} and V_1, those of the outflow tract of the right ventricle in HV_2 those of the anterior wall of the right ventricle in V_2 sometimes *in* V_3 those of the apical aspect of the anterior *wall of the* left *ventricle in* V_4 5 *and* basal aspect in HV_4 and HV_5 those *of the apical* portion of the *lateral* and posterolateral walls in V_5 and V_7 respectively those of the basal aspect in HV_6 and HV_7 those of the posterior wall *of* the left ventricle in V_8 and HV_8 those of the diaphragmatic surface *of the heart in* aV_F and the endocardial aspect in aV_R When *sufficient leads* are available to explore *these* aspects of the *heart* the interpretation of the *electrocardiogram* is simplified and expedited and errors are *minimized since* direct deductions can be made from recorded patterns instead of the *assumptions and inferences* necessary when the required leads have not been taken For these reasons *the extra* expense *for sensitized* paper and *slight* additional time on the part of the *technician* are *justifiable*

II PROCEDURE FOR THE INTERPRETATION OF A *TECHNICALLY SATISFACTORY* TRACING includes (A) determination of rate and rhythm (B) perusal of P *and* T_P waves to determine presence or absence of evidence *of abnormalities in auricular* activation *and* repolarization (C) study *of* the QRS *S T and T* waves in *each lead* to determine whether ventricular activation and repolarization *are normal* or abnormal and to diagnose *the* nature of any detected abnormalities *The experienced* observer more or *less* automatically *arrives* at *these* decisions in the course of a mere inspection of each *lead While* the necessary knowledge *and* experience are being acquired the development *of a* method *for* systematic survey of the tracing is

advisable to protect against errors from oversight The following procedure is recommended to those who have not worked out systems of their own

A DETERMINATION OF CARDIAC RHYTHM Intimate knowledge of the physiologic mechanisms and *clinical manifestations of the arrhythmias* is essential The electrocardiographic features are presented in detail in Section D organized according to a modification of the usual classification Electrocardiographers familiar with Section D recognize most arrhythmias from a mere inspection of the tracing and interpret the more difficult cases with the aid of calipers In the process of mastering the arrhythmias the following systematic approach differing from that of Section D may be of greater aid in the analysis of the tracing In this section the description of each arrhythmia is reduced to the bare minimum for the classification in Section D additional details are given

1 Identify the P and QRS deflections in each lead This usually can be accomplished at a glance Difficulties may arise when the P waves are of low voltage or when they are buried in the QRS-T complex The fact that P waves are oppositely directed in aV_R and and aV_F is of aid in identification of deflections of low voltage Even though obscure in all limb leads distinct P waves may be detectable in V_{3R} V_1 and HV_1 or in esophageal leads taken at the level of the atria In the presence of auriculoventricular dissociation P waves which fall between ventricular cycles should be clearly visible whereas those superimposed on the S-T segment or T wave are usually recognizable from the deformity in the S-T complex those recorded simultaneously with the QRS may cause visible notching but are often completely buried With the aid of dividers set at the interval between the closest pair of recognizable P waves the position of the buried P waves may be revealed and the nature of the auricular rhythm thereby established

2 Count the ventricular rate and if necessary the auricular rate

a Ventricular rate The method used depends upon whether rhythm is regular or irregular

1) Irregular rhythm The approximate rate may be determined by counting the number of QRS complexes within 30 consecutive large squares (6 sec) and multiplying by 10

2) Regular rhythm

a) Rate may be determined readily from the cycle length (R-R interval) by reference to the following table

R-R Interval			R-R Interval			R-R Interval		
Small Squares	Sec	Rate	Small Squares	Sec	Rate	Small Squares	Sec	Rate
30	1 20	50	23	92	65	16	64	94
29	1 16	52	22	88	68	15	60	100
28	1 12	54	21	84	71	14	56	107
27	1 08	56	20	80	75	13	52	115
26	1 04	58	19	76	79	12	48	125
25	1 00	60	18	72	83	11	44	136
24	96	62	17	68	88	10	40	150

b) Since 1500 consecutive small squares constitute a time interval of one minute the rate can be calculated by dividing 1500 by the number of small squares in the R-R interval

b Auricular rate If a QRS complex follows every P wave at a uniform interval the auricular rate will be the same as the ventricular If the relationship between the P and QRS waves varies in different cycles it will be necessary to determine the auricular rate in the same manner as described above for the ventricular rate

3 Determine whether the spacing of the P and QRS deflections is regular or irregular A glance at each lead will usually suffice for this decision and often for the recognition of the type of irregularity as well If uncertain by mere inspection of the electrocardiogram the nature of the irregularity in the P and/or QRS deflections should be determined with the aid of dividers A diagnosis is then established by integrating the data on the spacing of the P and QRS complexes with data on their interrelationships (heading 4) and respective contours (headings 5 and 6)

4 Note whether the relationship of the P waves and QRS complexes is constant or variable throughout the tracing Measure the P-R interval from the onset of the P to the onset of the QRS

a Constant relationship of P and QRS as indicated by a uniform P-R interval *in every* cycle of a given lead

1) Normal P-R interval The lower limit is 12 sec the upper limit depends upon age and heart rate In adults the upper limit of normal for the P-R interval is 21 sec at rates below 70 20 sec for rates between 70 and 90 19 sec for the 90 to 110 range 18 sec for rates between 110 and 130 and 17 sec for higher rates The upper limits of normal for children between 2 and 6 years of age are 04 sec below the adult figures for children between 7 and 13 the values are 03 sec below the adult and for adolescents the upper limit is 02 sec less than the adult Measurements slightly exceeding these figures have been obtained in persons without clinical or other electrocardiographic evidence of abnormalities

2) Prolonged P R delayed A-V conduction (first degree block) Every P wave is followed by a QRS at a P-R interval exceeding the upper limit of normal Before making a diagnosis of first degree block the possibility of a 2 1 block must be excluded by setting dividers at one-half of the P-P interval and placing one point of the dividers on a well-defined P wave and searching for an additional P wave in the portion of the tracing where the other point falls

3) Shortened P-R measuring less than 12 sec

a) Sinus rhythm

(1) Anomalous ventricular excitation through the bundle of Kent (Wolff-Parkinson-White syndrome) is recognized by the characteristic fusion QRS consisting of a broad slurred or notched premature component and a final component of normal contour

(2) Accelerated A-V conduction recognized by steeple-like P waves inverted in aV_R upright in aV_F a slightly shortened P-R interval and a QRS of normal contour

b) A V nodal rhythm recognized by upright P waves in aV_R and sharply inverted V-shaped P waves in aV_F associated with a short P R and a QRS of normal contour

b Variable relationship of P and QRS-T The entire tracing should be studied with the aid of dividers to determine (1) whether some but not all of the sinoauricular impulses reach and activate the ventricles to produce QRS-T complexes related to the preceding P waves or (2) whether none evokes a ventricular response with consequent independence of ventricular and auricular rhythms In the former event partial A V block must be differentiated from the relatively rare intermittent interference dissociation in the latter event complete A-V block and the rare complete interference

dissociation must be distinguished A-V block is characterized by partial or complete interruption of conduction through the A-V node with a resultant ventricular rate slower than the auricular Interference dissociation is an expression of a second more active pacemaker in the A-V node or ventricle that usurps ventricular control from the sinus node intermittently or continuously as a consequence the ventricles beat either at a more rapid rate than the auricles or at approximately the same rate but slightly in advance of the arrival of the sinoauricular impulse

1) Ventricular response to some but not to all auricular impulses

a) Partial A-V block The ventricles are still under auricular control but conduction is sufficiently impaired that some impulses fail to reach the ventricles resulting in dropped ventricular beats thus making the ventricular rate slower than the auricular The degree of partial A-V block is expressed as a ratio between the number of auricular and ventricular complexes and may be classified into 3 grades The electrocardiographic features of each form of partial A-V block are given in detail on page 95

(1) Low grade partial A-V block (second degree block) The ratio is subject to variation in different portions of the tracing ranging from 10 9 up to 3 2 related to fluctuations in vagal tone and sympathetic activity Electrocardiograms are further subdivided in accordance with the relationships of QRS to P in succeeding cycles

(a) Moebius type There is delayed A-V conduction with occasional dropped ventricular beats but an otherwise constant P-R interval

(b) Wenckebach type There is a progressive increase in the duration of the P-R interval in succeeding cycles until an auricular impulse fails to reach and activate the ventricles In the cycle following the dropped ventricular beat the P-R reaches its minimum (which may be within or above normal range) and in succeeding cycles progressive lengthening of the interval is repeated until another ventricular beat is dropped

(c) Paradoxical shortening of a prolonged P-R interval is a rare finding in partial A-V block and is characterized by significant shortening of the P-R interval from one cycle to the next succeeding beat described in more detail on page 96

(2) High grade partial A-V block with ratios of 2 1 or higher (3 1 4 1 etc) In 2 1 block the QRS follows every other P wave usually after a long normal or lengthened P-R interval The diagnosis is made by identification of the P wave with blocked ventricular response This P wave is detected by search for deformity in the preceding S-T segment or T wave and is identified by its shape and time relationships with other P waves as shown with the aid of dividers

(3) Very high-grade partial A-V block with atrioventricular conduction only when P waves fall within a critical time interval after the preceding QRS (usually from 45 to 75 sec after the onset of the preceding QRS) The differentiation from complete A-V block is made by the fact that P waves falling during this critical period are consistently followed by a QRS at a constant P-R interval

b) Intermittent interference dissociation complicating an escaped A-V nodal (or ventricular) rhythm with retrograde block The background for this unusual arrhythmia is (1) the presence of a pacemaker in the A-V node (or rarely in the ventricle) that generates impulses more rapidly than the sinus node (2) the preservation of forward conduction but block of retrograde conduction through

the A-V node The sinus node has exclusive control over the auricles as indicated by uniform P waves of sinus origin but competes with the A-V nodal pacemaker (or ventricular pacemaker) for control of the ventricles The electrocardiographic features will be elaborated on page 99 but in brief summary consist of (1) uniformly shaped and spaced P waves of sinus origin (2) a slightly faster escaped ventricular rhythm manifested by regularly spaced QRS complexes that make their appearance further and further ahead of the position expected if they had been of sinus origin (3) the periodic occurrence of a QRS sufficiently in advance to permit transmission of the next regular auricular impulse into the ventricles and elicitation of a response This interjects periodic cycles consisting of a P wave synchronous with the regular auricular rhythm followed after a P-R interval of 12 sec or more by a QRS that appears early in respect to those of independent rhythm originating from the lower pacemaker The decision as to whether the lower pacemaker lies in the A-V node or ventricles is made from the contour of the QRS-T during the escaped rhythm as compared with that during sinus control QRS complexes like those during sinus control indicate a nodal pacemaker broad and bizarre QRS complexes point to a ventricular pacemaker

2) Absence of ventricular response to any sinoauricular impulse The atria are under control of the sinus node the ventricles beat independently in response to an A-V nodal or idioventricular pacemaker This may be observed as a result of (a) complete block of antegrade conduction from auricles to ventricles as a consequence of which the ventricles beat at an independent rhythm slower than the auricular rhythm and fail to respond to any sinoauricular impulses regardless of the portion of the ventricular cycle in which the P wave falls (b) complete interference dissociation in which the ventricles beat at approximately the same rate as the auricles with QRS onset slightly in advance of the arrival of the sinoauricular impulse so that the latter reaches the *junctional* tissues when they are in a refractory state that prohibits conduction through the A-V node

a) Complete block of forward conduction from auricles to ventricles (third degree block) is almost always but not invariably accompanied by interruption of retro grade conduction from ventricles to auricles this necessitates subdivision into 2 categories

(1) Complete A-V block with interruption of both forward and retrograde conduction The ventricles beat at an independent rhythm slower than the auricles and fail to respond to any sinoauricular impulses regardless of the portion of the cycle in which the P wave falls The intervals between the QRS complexes are regular unless the ventricles are stimulated by more than one focus The shape of the QRS-T will depend upon the site of the ventricular pacemaker which may be classified as follows

(a) Pacemaker above bifurcation of Bundle of His The QRS-T complex is normal in duration unless there is complicating bundle branch block or intraventricular conduction defect

(b) Pacemaker below bifurcation of Bundle The QRS-T complex is broad and bizarre and is subject to changes in configuration as a result of a tendency for the idioventricular focus to shift in location The ventricular rate is generally slower than that prevailing when the pacemaker is above the bifurcation of the bundle and episodes of ventricular standstill are more common

(2) Complete forward block with preservation of retrograde conduction a rare phenomenon To make the diagnosis the presence of complete forward block

must be demonstrated first The preservation of retrograde conduction is established by the identification of intermittent oppositely directed P waves (i e upright in aV_R inverted in aV_F) following the QRS at a fixed interval

b) Complete interference dissociation complicating an escaped nodal (or ventricular) rhythm with retrograde block This rare arrhythmia requires (1) the presence of pacemaker in the A-V node (or rarely the ventricles) that generates impulses at approximately the same rate as the sinus node and sends them into the ventricles slightly in advance of the arrival of the sinoauricular impulse at the junctional tissue (2) the presence of a retrograde block The electrocardiographic features are described on page 109 in summary they consist of (1) regularly spaced P waves of sinus type (inverted in aV_R upright in aV_F) that occur either *immediately ahead of the QRS* (with P-R interval of less than 12 sec) superimposed upon it or immediately behind the QRS (2) independent regularly spaced QRS complexes that are usually normal in contour (nodal pacemaker) rarely bizarre (ventricular pacemaker or nodal pacemaker with abnormal intraventricular conduction) The relationship of P to QRS tends to vary slightly in different parts of the tracing due to the fact that rates of the two pacemakers are seldom absolutely regular

5 Note whether the P waves are uniform or variable in contour in each individual lead

a *Uniformity in contour of P waves in each individual lead indicates a single auricular* pacemaker The location of the pacemaker is determined from the shape and direction of the P waves particularly in Lead aV_R as contrasted with aV_F When the direction and contour of the P waves indicate that the rhythm is not of sinus origin rate is also taken into consideration in the differentiation between a passive A-V nodal rhythm and an active ectopic rhythm (i e supraventricular tachycardia and auricular flutter)

1) Sinus rhythm is indicated by uniform P waves that are inverted in aV_R and upright in aV_F occurring at a rate that is usually below 120 and almost invariably below 150

a) Regular sinus rhythm is present when cycle lengths do not differ by more than 12 sec

(1) Sinus bradycardia—rate below 60

(2) Sinus tachycardia—rate above 100

b) Uncomplicated sinus arrhythmia is present when cycle lengths differ by more than 12 sec but P waves and QRS-T complexes are uniform in contour and relationship in any given lead

(1) Phasic sinus arrhythmia is manifested by waxing and waning of cycle length related to respiration

(2) Nonphasic sinus arrhythmia—P waves are uniform in contour but P-P cycle lengths exhibit irregular variations apparently independent of breathing

c) Sinoauricular block complicating an otherwise regular sinus rhythm is manifested by intermittent pauses amounting to approximately 2 cycle lengths or multiples thereof during which there is absence of auricular and ventricular activity Further subdivision is made according to the character of the beat that terminates the block

(1) Sinoauricular block with resumption of sinus activity The beat terminating the block is characterized by a P wave P-R interval and QRS-T similar to that of other cycles

(2) Sinoauricular block with nodal (or ventricular) escape The P wave in the cycle terminating the block is either retrograde in type or not discernible because of burying in the QRS Source of the escaped beat is determined from the contour of the QRS T

(a) Nodal—QRS T similar to that produced by impulses of sinus origin

(b) Ventricular—QRS-T lengthened in duration and bizarre in contour

d) Coexistent sinus and A-V nodal rhythms with interference dissociation and retrograde block—discussed above

2) Passive A-V nodal rhythm with retrograde auricular conduction is manifested by evenly spaced uniform P waves that are upright in leads aV_R and aV_L and inverted and V shaped in aV_F The QRS complexes are normal unless there is a complicating ventricular lesion The rate should be below 100 in contradistinction to active nodal rhythms Further subdivision is made in accordance with the temporal relationships of the retrograde P and QRS

a) Upper nodal rhythm P wave precedes QRS usually by an interval of less than 12 sec but exceeding the latter in the presence of delayed A-V conduction

b) Middle nodal rhythm P wave is superimposed upon the QRS

c) Lower nodal rhythm P wave follows QRS after a variable interval depending upon the site of the pacemaker and the speed of retrograde conduction

(1) Reciprocal rhythm a rare phenomenon occurring in lower nodal rhythm and recognized by a second QRS following the retrograde P simulating the first QRS in contour and separated from it by an interval of 5 sec or less The probable mechanism is discussed on page 110

3) Active auricular ectopic rhythms

a) Supraventricular tachycardia is characterized by successions of rapid regularly spaced ectopic P waves occurring at rates usually between 150 and 220 In each lead the P waves are separated by intervals devoid of auricular activity The episode begins with a supraventricular premature beat and terminates with a pause sometimes followed by one or more escaped beats from another focus before resumption of sinus rhythm Supraventricular tachycardia is subdivided in accordance with

(1) The form of the P wave—2 varieties are distinguishable

(a) Auricular tachycardia The P waves are similar in direction to those of sinoauricular rhythm and bear close resemblance in respect to contour P R interval is 12 sec or greater and is often prolonged

(b) Nodal tachycardia The direction and form of the P wave are similar to those in passive A V nodal rhythm and rate may range down to as low as 100 The P waves may precede the QRS by an interval of less than 12 sec may be superimposed upon the QRS or may fall between the QRS and T depending upon the point of origin in the auriculoventricular node and the comparative speeds of retrograde conduction through the A-V node and auricles and antegrade conduction to the [illegible] es

(2) The [illegible] plexe[illegible] form [illegible] The QRS complexes [illegible] supraventricular in [illegible] bizarre in contour * due either to aberrant

*the P wave [illegible] complexes is differentiated by the fact that the

conduction through normal ventricles or to a complicating ventricular lesion causing defective intraventricular conduction The ventricles may respond to each auricular impulse (1 1 ratio) to every other impulse (2 1 ratio) or irregularly (variable block) rarely the ventricles may beat at a slow independent rhythm (complete A-V block)

b) Auricular flutter Auricular activity is manifested by regular uniform undulations that are continuous and without isoelectric interludes in at least some of the leads but not necessarily in all leads occurring at a rate generally between 250 and 360 The diphasic form of the undulations often bears a resemblance to saw teeth but sometimes only the troughs or crests of the waves can be made out because of obliteration of the remainder of the undulation by superimposed QRS and T waves The ventricles most commonly respond regularly to each second or fourth undulation (2 1 to 4 1 ratio) rarely the ventricles are regular at some other ratio or because of complete A-V block Irregular spacing of the QRS is a common ventricular response to auricular flutter and results in variable ratios which usually fluctuate between 2 1 and 4 1 The QRS-T is usually not altered with 4 1 ratios but may become aberrant with 2 1 ratios or faster ventricular rates

b Variability in the contour of the P waves in any given lead is generally attributable to more than one focus for auricular excitation but may result from variability in intra-atrial conduction of impulses originating in the sinus node The first problem is to determine whether the findings are referable to some complication of an underlying sinus rhythm or to an irregular ectopic rhythm such as auricular fibrillation In the former event the next problem is to determine whether the P wave variations are explainable by a shift in the point of exit of the impulse from the sinus node or a change in its intra-auricular pathway or whether they indicate the presence of one or more ectopic foci displacing the sinus node

1) Sinus arrhythmia with shifting sinus pacemaker The wandering of the pacemaker within the sinus node is recognized by slight variations in the amplitude and contour of the P waves and in the duration of the P-R interval in the presence of sinus arrhythmia The P waves tend to be taller and more sharply peaked in the shorter cycles lower and more rounded in the longer cycles

2) Sinus rhythm (or sinus arrhythmia) complicated by ectopic P waves coming either from an auricular or A-V nodal focus or rarely from a ventricular focus by retrograde conduction The approximate location of the focus is determined from the direction of the P wave in the unipolar limb leads When the focus is located in the right atrium the P wave is inverted in Lead aV_R but differs significantly in contour and amplitude from the P wave of sinus origin it is usually upright in aV_L and aV_F but may be shallowly inverted in the former when the focus is in the upper part of the right atrium or shallowly inverted in the latter when the focus is in the lower portion P waves arising from the left atrium are upright in aV_R if the focus is high in the atrium they are inverted in aV_L and upright in aV_F if the ectopic site is low they may be reversed in direction in the 2 latter leads When the impulse originates from within or near the A-V node the P wave is sharply inverted and V-shaped in aV_F and upright in aV_R and aV_L and the P-R interval is shorter than in the sinus beats generally less than 12 sec The nature of the arrhythmia is determined from the temporal relations of the ectopic beats to the sinus rhythm

a) Sinus rhythm complicated by premature auricular or nodal beats The diagnosis is made by the identification of a P wave that is (1) premature as shown by its occurrence in advance of the next anticipated P wave (2) ectopic in origin as indicated by significant differences in direction and/or contour from P waves of

sinus origin Premature ectopic beats generally occur singly occasionally in short runs a single premature beat or the first in a series shows a constant time relation to the preceding P and QRS-T complexes Judgment of contour or even recognition is often complicated by the tendency for premature P waves to be superimposed upon the T waves of the preceding cycle, in which event they may be manifested as a notch or may be perfectly fused with the T wave. Supraventricular premature beats may be further classified in accordance with the location of the ectopic focus the time required for a ventricular response the contour of the QRS T

(1) Location of the ectopic focus (i e whether in right atrium left atrium or A-V node) is determined from criteria given above The presence of 2 or more ectopic foci is indicated by significant differences in the contour and/or direction of the ectopic P waves in a given lead

(2) P-R interval

(a) Auricular premature beats The P-R interval may be similar to that of the associated sinus beats or slightly longer or shorter depending in part on the location of the ectopic focus, on the degree of prematurity and on the conductivity of the A-V node When very premature the P-R interval may be significantly prolonged or a QRS response may fail to occur, due to partial or complete refractoriness of the junctional tissue

(b) Nodal premature beats The P-R interval depends upon the portion of the node in which the focus is located and the relative speeds of retrograde and forward conduction If the focus is in the upper portion of the node the aberrant P may precede the QRS by an interval of 02 to 12 sec If lower down the aberrant P wave may be submerged in the QRS or may follow it In the event of retrograde block a P wave of sinus origin may be detectable just before in or just after the QRS the nodal origin for the latter being indicated by a contour like that of the QRS-T complexes occurring in response to a sinoauricular impulse but premature in time

(3) QRS T complex following a supraventricular ectopic beat may be identical with that due to an impulse originating in the sinus node or it may differ very slightly in height shape or duration When the P wave is quite premature, the impulse may be blocked or it may take an aberrant course through the ventricles resulting in a bizarre QRS-T complex resembling that of premature ventricular beats For this reason it is necessary to search for a premature P wave before deciding whether a premature QRS-T is arising from an ectopic auricular or a ventricular focus

b) Sinus [illegible]
lar) l [illegible]
T-P [illegible]
still [illegible] against prolonged cardiac standstill [illegible] pause may occur during the course of marked sinus arrhythmia as a result of sinoauricular or partial A-V block or following a ventricular premature beat

Escaped beats are recognized as nodal in origin when the pause is terminated by a QRS-T complex that is identical with or only slightly different from the QRS T complexes resulting from a sinoauricular impulse and as ventricular in origin when the pause is terminated by a broad bizarre QRS quite different from that occurring in response to sinus impulses

Escaped nodal beats are further characterized by the type of auricular response ( ) retrograde conduction is indicated by a P wave that is upright in aV_R inverted in aV_F and immediately precedes coincides with or follows the

QRS (2) retrograde block may be presumed when no evidence of auricular activity is detectable (3) interference is indicated when a P wave similar to that of sinus beats is found just ahead of the QRS (at a P-R interval less than 12 sec) superimposed upon the QRS *or following it* *If the* sinus node recovers in time to gain control over the whole heart at the next cycle the previous beat is referred to as an escaped nodal beat with isolated interference If the nodal center retains control over the ventricles for several consecutive cycles while the sinus node controls the auricles the phenomenon is designated as an escaped nodal rhythm with interference dissociation *(page 109)* *If the associated* normal QRS-T complexes and retrograde P waves continue for several cycles the rhythm is designated as an escaped nodal rhythm with retrograde auricular conduction

c) Wandering pacemaker between the sinus and A-V nodes There is a single pacemaker for the auricles and ventricles which shifts back and forth between the S-A and A-V nodes as a result of vagal depression of the more irritable focus P waves of sinus origin (inverted in aV_R upright in aV_F) are replaced during the course of a few cycles by oppositely directed retrograde P waves and abnormally short P-R intervals of nodal origin During the transition fusion P waves *intermediate between those of sinus and nodal origins are prone to occur* as the result of simultaneous auricular activation by impulses from the S-A and A-V nodes When the pacemaker wanders back to the sinus node the reverse changes *in P waves and P-R intervals occur*

d) Auricular parasystole This is a rare disorder manifested by two independent sets of P waves one of sinus origin the other arising from an isolated ectopic focus distinguished by the following characteristics (1) a varying temporal relationship to the preceding cycle of sinus origin as contrasted to the constant relationship characteristic of repeated auricular premature beats from a single focus (2) a constant but slower rhythm than that of sinus origin which is best demonstrated with the aid of dividers *Gaps of an exact multiple of the para*systolic interval may occur when the ectopic focus happens to discharge at a time when the auricles are responding to the sinus impulse Likewise gaps in the sinus rhythm may occur when the sinus node discharges during or shortly after an auricular response to the parasystolic focus

3) Auricular fibrillation In place of discrete P waves there are rapid auricular undulations of varying size contour and spacing occurring at rates generally between 400 and 600 sometimes of such low voltage to be demonstrable only in right precordial or esophageal leads A totally irregular ventricular response* *is elicited and is subject to great variations in rate* depending upon the degree of functional block *in the A-V node and may be as high as 200 to as low as 50* When all *of the* QRS-T complexes are derived from impulses transmitted through the A-V node they are usually but not necessarily uniform in shape in any given lead An impulse transmitted from the fibrillating auricles through the A-V node may reach the ventricles before they have fully recovered from the previous beat and consequently may spread through the ventricles in an aberrant fashion to give rise to a bizarre QRS-T complex following closely upon a normal QRS-T and resembling a ventricular premature beat Intermittent bizarre QRS-T complexes may also be the result of *intermittent ventricular* premature beats complicating auricular fibrillation A definite diagnosis of ventricular premature beats is justifiable only when there is fixed coupling

*An exception to the totally irregular ventricular rhythm occurs in the presence of a complicating complete A V *block the* existence of auricular fibrillation is established by the irregular auricular undulations and the complete A V block by the perfectly regular ventricular rhythm

6 Note whether the QRS-T complexes are uniform or variable in contour in each individual lead

a Uniformity in contour of QRS-T complexes in each individual lead indicates a single pacemaker for ventricular activation The relationship of the QRS to the P waves serves as a basis for determining whether (1) the impulse responsible for ventricular activation originates within the ventricle at some point below the bifurcation of the Bundle of His (2) the impulse is transmitted from auricle to ventricle through an anomalous pathway (Bundle of Kent) (3) the impulse reaches the ventricles through the A-V node In the latter event the duration and contour of the QRS serve as an index as to whether the impulse spreads through the ventricles in a normal or abnormal fashion

1) Unifocal ectopic ventricular rhythms are characterized by broad bizarre QRS complexes with oppositely directed T waves completely independent of all antegrade impulses arising above the bifurcation of the Bundle of His as shown by the fact that the P waves fall at different points in reference to the QRS-T and occur at a rate mathematically unrelated to the ventricular rate Although the demonstration of the absence of ventricular response to impulses of auricular origin is essential to the diagnosis of an ectopic ventricular rhythm conduction in the reverse direction is compatible and is occasionally observed resulting in retrograde P waves following each QRS of every second third or fourth ventricular complex Ectopic ventricular rhythms may be subdivided into 2 groups (a) passive manifested by a slow ventricular rate and occurring because of complete A-V block or arrest of impulse formation in higher centers and (b) active manifested by a rapid ventricular rate Although the following ectopic ventricular rhythms are classified under the heading of uniformity in QRS-T contour they do not always comply with this characterization variability in QRS-T contour may be observed as a result of change in the manner of spread through the ventricles as well as from superimposition of P waves on different portions of the QRS-T complexes

a) Passive ectopic rhythms Idioventricular rhythms associated with complete A-V block or escaping as a result of depressed impulse formation in the S-A and A-V nodes have been described on pages 108-110

b) Active ectopic rhythms

(1) Unifocal ventricular tachycardia is characterized by paroxysms during which the ventricular rate may range from 120 to 250 but is generally above 170 The paroxysm begins abruptly with a ventricular premature beat and terminates with a pause followed by resumption of sinus (or nodal) rhythm During the paroxysm the ventricular rhythm may be perfectly regular or may show slight irregularities in cycle length The QRS complexes are prolonged in duration and coarsely slurred or notched the T waves are readily demarcated and are opposite in direction to the main deflection of the QRS The approximate location of the focus is determined from the QRS patterns in multiple precordial leads as described on page 106 Ventricular tachycardia is differentiated from auricular tachycardia complicated by bundle branch block by demonstrating an independent auricular rhythm with P waves falling at variable points in reference to the QRS-T or by showing retrograde auricular activation and at the same time excluding forward conduction from auricles to ventricles

(2) Ventricular flutter In place of a clearly definable QRS complex and T wave the ventricular deflections consist of rapid uniform regular undulations (i e over 250 per minute) in which QRS and T components are blended and inseparable If auricular activity is discernible in the tracing the P waves are independent of the ventricular undulations

2) Anomalous ventricular excitation through the Bundle of Kent a neuromuscular bundle found in a few human hearts connecting the lateral aspect of the right atrium and surface of the right ventricle and/or the posterior aspect of the left atrium and ventricle The bundle of Kent may function intermittently transmitting the sino-auricular impulse to the ventricular terminus in advance of the arrival through the Bundle of His producing the Wolff-Parkinson-White syndrome, characterized by

a) Normal P waves of sinus origin

b) Shortening of the P-R interval below 12 sec as a result of premature onset of the QRS

c) A QRS deformity characterized by a fusion complex consisting of a coarsely slurred or notched premature component and a smooth thin imaged final component The fusion of the coarsely slurred or notched premature component with the normal final component results in (1) a slurred or notched QS deflection in the lead or leads facing towards the Bundle of Kent (right precordial leads in the presence of a right lateral bundle back leads with a posterior bundle) and (2) slurred or notched R waves in leads over the opposite walls The T wave tends to be opposite to the major deflection of the QRS The greater the disparity in time of arrival of the impulse transmitted through the 2 pathways the greater the amount of ventricular myocardium activated by the anomalous route the greater the amplitude and duration of the premature component and the greater the deformity of the QRS and T waves

d) Lengthening of the QRS interval at the expense of the P-R segment without change in the interval between onset of P and end of QRS

e) A tendency towards paroxysms of tachycardia The episodes of tachycardia are usually characterized by P-R intervals and QRS-T complexes indicative of transmission through the A-V node rarely by the WPW pattern

3) Ventricular activation through the A-V node and Bundle of His is established by a study of the relationship of QRS to P as described in detail above in sections 4 and 5 For example in sinus rhythm a constant P-R interval of 12 sec or more indicates ventricular activation by way of the A-V node in auricular fibrillation total irregularity in spacing together with uniformity in QRS contour in a given lead indicates a supraventricular control of the ventricles After establishing that ventricular activation resulted from impulses transmitted through the A-V node the decision as to whether conduction of the activating impulse through the ventricles is normal or abnormal is made from the duration and contour of the QRS

a) Normal intraventricular conduction is indicated by QRS complexes 10 sec or less in duration and normal in contour in all leads

b) Defective intraventricular conduction is indicated by a QRS interval of 12 sec or more together with abnormal slurring or notching lesser degrees may be manifested by abnormal slurring or notching in association with a QRS interval of 09 to 11 sec

(1) The ventricular site of the conduction defect is determined from the chest leads exhibiting an abnormally late onset of the intrinsicoid deflection (i e the precipitous final downstroke which begins at the peak of the R (or R') wave) Late intrinsicoid deflections are not followed by S waves

(a) Left ventricle The late intrinsicoid deflection occurs in leads facing the epicardial surface of the left ventricle (V_8 V_7 almost always V_6 usually V_5) In the severer grades of conduction defect the intrinsicoid deflection in these leads begins 08 sec or more after the onset of the QRS in the lesser grades 06 to 08 sec

(b) Right ventricle The late intrinsicoid deflection occurs in leads facing the epicardial surface of the tricuspid ring (V_{4R} V_{3R} usually V_1), and/or conus pulmonalis (HV_2) In the severer grades it begins 08 sec or more after the onset of the QRS in lesser grades from 05 to 07 sec

(2) The nature of the conduction defect is determined from the direction of the initial deflection of the QRS and the relative duration and amplitude of each of its component phases

(a) Left ventricular conduction defects

(1)) Left bundle branch block is manifested by an initial upstroke in all leads facing the epicardial surface of the left ventricle due to early postivity of the left ventricular cavity The left bundle branch block is classed as complete when QRS duration is 12 sec or more incomplete when 10 to 11 sec

(2)) Outer wall of the left ventricle is the site of the conduction defect when the leads with a late intrinsicoid deflection have a distinct q wave reflecting initial negativity of the left ventricular cavity and indicating septal activation in the usual left to right direction When the q wave is normal (i e duration of 02 sec or less amplitude less than 25% of the R) whereas the ascending limb of the R is tall and prolonged the increased time interval is referable to prolongation of conduction through the outer wall of the left ventricle usually the result of left ventricular hypertrophy When the Q wave is abnormal (i e duration of 03 sec or more amplitude more than 25% of that of the succeeding R) the delayed conduction is attributable to infarction of the subendocardial layer of the subjacent myocardium

(b) Right ventricular conduction defects

(1)) Initial R wave (and subsequent R') deflection in right ventricular leads with late intrinsicoid deflection indicates uncomplicated right bundle branch block The lesion is complete when QRS duration is 12 sec or more incomplete when 09 to 11 sec

(2)) Initial Q wave in right ventricular leads with late intrinsicoid deflection results in a pattern that may be due to

(a)) Septal infarction with right bundle branch block established by characteristic S-T changes and by evidence of continuation of the infarct into the anterior wall of the left ventricle

(b)) Marked right ventricular hypertrophy with delayed conduction in the free wall of the right ventricle

b Variability in contour of the QRS-T complexes in any given lead It is necessary to determine whether there is (1) an exclusive supraventricular pacemaker with variability in intraventricular conduction (2) one or more ectopic ventricular foci in addition to a supraventricular pacemaker (3) an ectopic ventricular rhythm from two or more foci

1) Exclusive supraventricular pacemaker The means of arriving at this conclusion have been described Under these circumstances variations in QRS contour in a given lead may be due to

a) Intermittent Wolff-Parkinson White syndrome with alternation between normal QRS complexes produced by impulses transmitted exclusively through the A-V node and fusion complexes produced by impulses transmitted partly through

the Bundle of Kent partly through the Bundle of His The diagnostic characteristics of the latter have been described There is one additional criterion of value when the syndrome is intermittent namely that the time from onset of P to the S-T junction (P-J interval) is the same in the fusion complexes as in the normal

b) Intermittent bundle branch block is established by the demonstration of alternation between QRS complexes of normal duration and of bundle branch block type in the presence of a perfectly constant P-R interval In some cases the P-P interval of the cycles exhibiting normal QRS duration is significantly longer than that of the cycles exhibiting bundle branch block The extra time presumably permits recovery from fatigue that interfered with conduction in the bundle branch Intermittent bundle branch block may be associated with a constant rate and may still reflect fatigue since a conduction defect in alternate cycles provides twice the usual period for recovery of a fatigued bundle

c) Respiratory alterations in cardiac position may cause considerable cyclic fluctuation in QRS contour in leads at the transitional zone without change in P-R interval or in QRS duration

2) Supraventricular pacemaker (i e sinus rhythm nodal rhythm or ectopic auricular rhythm) complicated by intermittent beats from one or more ectopic ventricular foci

a) Underlying supraventricular rhythm complicated by premature ventricular beats The diagnosis is made by the demonstration of a premature bizarre QRS-T complex *together with the exclusion of a premature ectopic P wave* The QRS complexes of premature ventricular beats are invariably longer in duration and different in shape from those of supraventricular origin They usually exceed 11 sec in duration and are coarsely slurred or notched The T wave is opposite in direction to the main deflection of the QRS Ventricular premature beats may be further classified in accordance with source frequency time relationships and effect on auricular and ventricular rhythm

(1) Number and location of foci

(a) Single focus is indicated by uniformity in contour of all premature beats in each individual lead If the premature beats are frequent the approximate location of the focus can be determined from the contour in multiple thoracic leads A QS complex is recorded in the lead or leads overlying the epicardial surface of the ventricular segment from which the premature beat is originating a monophasic R wave is registered in leads from the opposite wall of the heart a diphasic RS complex is recorded in intervening leads The upright component of this RS complex is small and brief in duration in leads over portions of the ventricle near the ectopic focus and increases in amplitude and duration at the expense of the S wave in leads farther and farther removed from the focus

(b) More than one focus is present when two or more premature beats in the same lead show decided differences in direction and/or contour

(2) Frequency Ventricular premature beats may be occasional or frequent may occur singly in pairs or in short runs They may follow each supraventricular beat resulting in a bigeminal rhythm or each second supra ventricular beat causing a trigeminal rhythm

(3) Relation to preceding ventricular cycle All ventricular premature beats that come from the same focus and occur singly have a constant temporal relation to the antecedent ventricular cycle The time of predilection for

ventricular premature beats is early in diastole superimposed on the U wave of the preceding cycle Another common finding is a late ventricular premature beat superimposed on the P wave or following it by an interval significantly shorter than the customary P-R

(4) Auricular rhythm is generally undisturbed the sinoauricular P wave occurs at the scheduled time and is sometimes recognized as a notch on the QRS-T and is at other times detectable only by the demonstration of an undis urbed auricular rhythm with the aid of dividers When the ventricular beat is very premature and the heart rate slow a retrograde impulse may pass from the ventricles through the A-V node and excite the auricles before the sinus node is discharged producing a retrograde P wave (inverted in aV_F upright in aV_R) in advance of the scheduled time for the sinus P wave

(5) Ventricular rhythm Provided that the auricles are beating regularly under sinus control the interval between the ectopic and next supraventricular QRS is lengthened sufficiently to compensate exactly for the shortening of the preceding cycle The pause is noncompensatory under the following circumstances

(a) Retrograde auricular systole

(b) Interpolation of the ectopic ventricular beat between two normally spaced QRS complexes This occurs when a premature ventricular beat is blocked from further retrograde conduction at the A-V node and when the heart is so slow that the ventricles recover in time to respond to the *next impulse* coming down from the auricle The cycle which follows an interpolated extrasystole is likely to show a prolonged P-R interval and a distorted QRS-T This is the only type of premature ventricular beat which is a true extrasystole

(c) Underlying ectopic auricular rhythm In auricular fibrillation complicated by ectopic ventricular beats the ventricular rhythm remains totally irregular Differentiation between ectopic ventricular beats and aberrant intraventricular conduction of supraventricular impulses is difficult unless the bizarre QRS-T complexes are frequent If of ectopic ventricular origin they will show a constant coupling to the antecedent ventricular complex if due to aberrant intraventricular conduction of a supraventricular impulse the relation to the antecedent ventricular complex will be variable

b) Underlying sinus rhythm complicated by escaped ventricular beats The diagnosis is made by the demonstration of a bizarre QRS-T complex without associated P wave occurring after an interval longer than the customary R-R interval

c) Ventricular parasystole complicating sinus rhythm is suspected when ectopic complexes resembling ventricular premature beats fall at different points in relation to the QRS-T complexes of sinus origin The diagnosis is verified by demonstrating that the interval between any 2 ectopic complexes that are separated by cycles of sinus origin is a multiple of the interval between 2 contiguous ectopic complexes

3) Multifocal ectopic ventricular rhythms are characterized by irregularly spaced broad bizarre QRS complexes of variable contour with oppositely directed or blended T waves The ventricular complexes are completely independent of all antegrade impulses arising above the Bundle of His They may be classified as follows

a) Multifocal passive ectopic rhythms In complete A-V block more than one idioventricular pacemaker may be present giving rise to QRS-T complexes in a given lead that differ significantly in direction and/or contour

b) Active ectopic rhythms

(1) Multifocal ventricular tachycardia differs from the unifocal variety in that ectopic QRS-T complexes of two or more distinctive types are present in a given lead Each type of QRS complex is independent of auricular activity and is prolonged in duration coarsely slurred or notched and followed by a readily demarcated T wave of opposite direction The most common finding is an alternation between two different types of QRS-T patterns

(2) Ventricular fibrillation is characterized by irregular undulations of varying size and shape without demarcation between QRS and T

B PROCEDURE FOR THE INTERPRETATION OF THE AURICULAR COMPLEX This consists of the P wave representing auricular activation and the T_p wave representing auricular repolarization The utilization of P waves in the analysis of cardiac rhythm has been discussed in detail If sinus rhythm is present the study of the P waves should be extended to provide (1) estimation of anatomic relations of the atria to the various electrode positions (2) evaluation of auricular activation and repolarization

1 Estimation of anatomic relations of the atria to the various electrode positions is based on the direction and contour of the P waves The following inferences can be drawn provided sinus rhythm is present

a Multiple precordial leads The approximate position of the right atrium in reference to the anterior chest wall may be ascertained if sufficient leads are taken from the right precordium The P wave is inverted when the electrode is above and to the right of the atrium, it is diphasic with steep intrinsicoid downstroke when the electrode is over the atrium it is upright with precipitous descending limb when the electrode is in the vicinity of the right atrioventricular groove and upright with gently sloping limbs when the electrode is over either ventricle This information is of indirect aid in the interpretation of the ventricular complex

b Esophageal leads The relation of the electrode to the atria is determined from the contour of the P wave When the electrode is behind the atrium, the P wave is characteristically diphasic and consists of an initial upward component analogous to the R wave of the ventricular complex followed by a precipitous downstroke or intrinsicoid deflection which generally continues downward below the isoelectric line as a negative component analogous to the S wave In esophageal leads opposite the atrioventricular groove there is a tall upright P wave with steep intrinsicoid downstroke in esophageal leads at the level of the upper margin of the left atrium there is a reciprocal P wave When the electrode is well above or below the atrial level the P wave is monophasic and does not exhibit an intrinsicoid deflection In high esophageal leads the P wave is inverted with smooth rounded limbs In leads from the stomach and lower esophagus opposite the ventricle the P wave is upright and has gently sloping limbs like those in V_5 and V_6

c Unipolar limb leads In the presence of sinus rhythm the P wave in Lead aV_R is always inverted whereas that in aV_F is always upright The direction of the P wave in AV_L is subject to considerable variation in different patients depending upon the position of the heart in the chest Inverted or diphasic P waves in aV_L indicate that the heart is in a position that will facilitate transmission of potential variations of the ventricular cavities through the atrioventricular orifices to the left arm hence the presence of inverted or diphasic P waves in aV_L consitutes a warning against attaching pathologic significance to Q waves in this lead On the other hand the registration

in aV_L of an upright P wave with gently sloping limbs like those in V_5 V_6 suggests predominant transmission of auricular potential variations from the lower portion of the left atrium through the ventricular wall to the left arm in a manner analogous to their transmission to left axillary positions Under these circumstances the QRS-T pattern in aV_L should be representative of the potential variations of epicardial surface of the ventricle

2 Evaluation of auricular activation and repolarization

a Auricular activation is manifested by the P wave The decision as to whether auricular activation is normal or abnormal is made from the duration and contour of the P wave and from its maximal amplitude in the customary precordial and limb leads

1) Duration and contour of the P wave Duration is determined in the precordial or limb lead where the P wave appears to be widest but care must be taken to avoid inclusion of the T_P wave Since it is customary to demarcate time intervals on convex surfaces measurements of the duration of an upright P wave are made from the point on the lower margin of the baseline where the deflection begins its upward curve to the corresponding point where it returns to join the baseline whereas measurements of the duration of an inverted P wave are made from points on the upper margin of the baseline Contour is noted in each lead

a) Normal findings The duration of the P wave should fall within a range of 06 to 11 sec averaging 09 sec The P wave is usually smooth in contour but exhibits fine notching in approximately one-third of normal cases This notching is prone to occur near the summit and is more common on the upstroke than on the downstroke It is of no significance provided that the duration of the P wave is less than 12 sec Rarely a P wave duration of 12 sec has been found in young adults with clinically normal hearts This finding should not be classed as a normal variant unless the P wave is smooth in contour and the electrocardiogram negative in other respects

b) Abnormalities A P wave duration of 12 sec should be regarded as abnormal except under the circumstances given above a measurement exceeding 12 sec is always abnormal Prolongation of the P wave is generally accompanied by coarse notching and is indicative of defective intra-atrial conduction The etiology of the defect must be determined clinically since the electrocardiographic findings are nonspecific

2) Amplitude of the P wave is measured from the top of the isoelectric line to the summit of upright P waves and from the bottom of the isoelectric line to the nadir of inverted P waves

a) Normal findings The P wave should not exceed 2 5 mm in amplitude in the unipolar limb leads or in precordial Leads V_1 through V_6 It seldom exceeds 1 mm in precordial leads overlying the ventricles Tall pointed but narrow church-steeple-like P waves are recorded in lead aV_F in vertical position of the heart They are recognized as a normal variant when their duration is below 12 sec

b) Auricular hypertrophy is characterized by (1) P wave amplitude of 3 mm or more (3 mv) in at least one of the unipolar limb or customary precordial leads (2) P wave duration of 12 sec or greater [illegible] accompanied by coarse notching (3) [illegible] right atr[illegible] Hypertrophy of the the right [illegible] groove hypertrophy of the left atrium tends to exaggerate and prolong the negative phase of the P wave in such leads near [illegible] the P wave in leads near and to increase the amplitude of the P wave in esophageal leads

b <u>Auricular repolarization</u> results in the T_p wave, which begins immediately after the P wave and lasts for a total of 22 to 26 sec The level of the T_p deflection should be determined with reference to the T-P segment and the comparative directions of the P wave and T_p wave noted An erroneous impression as to depth of the T_p wave may be obtained in the presence of tachycardia sufficient to cause superimposition of the P wave on the preceding T and thus obliterate the T-P segment

1) <u>Normal findings</u> When visible, the T_p wave is opposite to the P and hence is normally directed downward in precordial leads and aV_F upward in aV_R In many cases the T_p wave is undetectable because of its low voltage and because a considerable portion is covered by the QRS when the P-R interval is normal The displacement from the T-P segment usually amounts to 5 mm or less, but may range up to 1 0 mm or rarely more In the latter event the T_p wave may cause pseudo depression of the S-T junction below the T-P segment a situation recognized from the presence of a corresponding depression in the interval between the end of the P and the onset of the QRS

2) <u>Abnormalities</u>

a) <u>Auricular hypertrophy</u> The T_p wave is exaggerated in amplitude and opposite in direction to the P wave

b) <u>Auricular infarction</u> The T_p wave may be displaced in the same direction as the P wave during acute injury and may show serial changes during organization A-V block facilitates recognition of this abnormality

C PROCEDURE FOR THE INTERPRETATION OF THE VENTRICULAR COMPLEX This consists of the QRS produced by ventricular activation the S-T junction marking the end of activation and the S-T segment and T wave produced by repolarization

1 <u>Multiple thoracic leads</u>

a <u>Duration of the QRS</u> representing the time required for ventricular activation must be determined in leads where the onset and termination of the complex are clear-cut All measurements of time are made in a horizontal plane from onset to end of the complex A lead is chosen in which the RS amplitude is relatively large and in which the QRS interval appears to be longest these stipulations are made to avoid selection of a lead in which either the first or last part of activation is isoelectric and thus not recorded Usually a precordial lead just to the left or just to the right of the transitional zone proves most suitable Measurements are made with the aid of a hand lens from the point where the furthermost margin of the baseline starts curving convexly to form the initial deflection of the QRS (i e the top of the baseline in the presence of a Q wave the bottom in the presence of an initial R wave) to the point where the string shadow suddenly thickens to resume the baseline (i e the top of the baseline if an S-T junction the bottom if an R-T junction) Estimations made with a hand lens are dependable within a range of ± 005 sec

b <u>Duration of the Q-T interval</u> representing the total time required for both ventricular activation and repolarization must be determined in a lead where the onset of the QRS and end of the T wave are clear-cut Care should be exercised to differentiate the U wave and to terminate the measurement at the end of the T wave

c <u>Localization of the transitional</u> ... the potential <u>variations of the epicardial sur</u> ... If a sufficient number of leads has b... ast should be evident in the QRS patterns recorded from the right precordium and those from the left axilla or back a transition should be demonstrable in leads from inter-

vening points If the QRS patterns in leads at the right and left ends of the series are roughly the reciprocal of one another the transitional zone should be located and the leads dominated by the potential variations of each ventricle should be identified If not sufficient additional leads should be taken to attain this objective Adequate coverage will almost invariably be provided by Leads V_{3R}-V_8 inclusive HV_1 - HV_8 inclusive and aV_R aV_L and aV_F which are recommended as a routine for the first electrocardiographic study (page 16) When a repeat electrocardiogram is requested it is customary to take V_1-V_8 inclusive and aV_F unless the original tracing revealed the desirability of other leads Leads from points to the right of the transitional zone reflect principally the potential variations of the right side of the septum and epicardial surface of the right ventricle or right atrium depending upon anatomic position, as judged from P waves as described on page 30 and are for the sake of convenience designated as right ventricular leads leads from points to the left of the transitional zone reflect principally the potential variations of the left side of the septum and the epicardial surface of the left ventricle and are referred to as left ventricular leads

1) Transitional zone

a) Location The transitional zone is situated between the lead farthest to the left with R/S relationships comparable to those in right ventricular leads and the lead farthest to the right with R/S relationships like those in left ventricular leads If found to the left of position V_4 an effort should be made to determine whether the displacement is attributable to a mere clockwise rotation to right ventricular enlargement or to both The transitional zone in the high precordial leads often does not correspond with that in the customary precordial leads and therefore must be located separately The width of the transitional zone will depend upon the anatomic relationships of the anterior terminus of the interventricular septum with the precordial positions An abrupt transition between 2 successive precordial leads is found when the anterior end of the septum is more or less perpendicular to a line joining the precordial electrode positions a gradual transition extending over 2 to 3 leads may be found when the apex is displaced backward or when the pathway of the electrode is parallel to the septum

b) Characteristics of the QRS T pattern in leads from the transitional zone

(1) QRS pattern depends upon the position of the electrode with reference to the septum when the heart is in diastole When the electrode position is to one side of the septum the QRS complex tends to resemble that in other leads over the same ventricle when the electrode straddles the septum the transitional zone is marked by a slurred or notched complex of relatively low voltage composed of an R and S deflection of approximately equal amplitude The notches are synchronous with R waves recorded in leads to the right or left of the transitional zone Bizarre QRS patterns at the transitional zone not classifiable into one of the foregoing groups suggest a myocardial lesion but must be interpreted in the light of the findings in adjacent right and left ventricular leads

(2) T pattern depends upon the position of the electrode with reference to the septum when the heart is in systole The transitional zone for the T wave does not necessarily correspond with that for the QRS due to the fact that mechanical systole begins after the inscription of the qR deflection and continues during the registration of the T wave The counterclockwise mechanical rotation usually associated with systole may sufficiently alter relationships of the heart to electrodes in the vicinity of the septum to shift the transitional zone for the T wave to the right of that for the QRS This becomes of importance in abnormal electrocardiograms in which the S-T segments and T waves in leads over-

one ventricle are opposite in direction to those over the other ventricle Thus a QRS pattern like that in right ventricular leads may be associated with a T wave like that in left ventricular leads or with a fusion S-T complex the first portion of which is chiefly right the terminal portion chiefly left ventricular in origin

2) Identification of leads reflecting principally the potential variations of the right ventricle These leads are situated to the right of the transitional zone The relationship of the electrode to the right atrium and ventricle respectively is judged from the contour of the P wave as described on page 30 A minimum of one lead near the tricuspid ring or inflow tract (V_1 or V_{3R}) one over the anterior wall of the right ventricle and one in the vicinity of the conus pulmonalis or outflow tract (HV_2) is necessary

3) Identification of leads reflecting principally the potential variations of the left ventricle These leads are situated to the left of the transitional zone A minimum of one lead facing the anterior one facing the lateral and one facing the posterior wall at the level of the apex (V_4-V_8) and at the level of the base (HV_4-HV_8) is necessary

d Configuration of the ventricular complex in the various leads reflecting principally the potential variations of the epicardial surface of the right ventricle as contrasted with the configuration in the various left ventricular leads With especial attention to the features given below the QRS and S-T patterns in the various right ventricular leads should be studied individually and compared with one another then a similar analysis should be made of the QRS and S-T patterns in the various left ventricular leads and finally the findings in the former and latter should be contrasted In succeeding descriptions when the relative amplitude of two or more phases of the QRS is significant a lower case letter will be used to indicate a deflection of relatively small amplitude and short duration an upper case letter for a deflection of relatively large amplitude and duration When both deflections of a diphasic complex are approximately equal capital letters will be employed for each when the number of phases and relative amplitudes are not pertinent the capital letters QRS will be used regardless of the configuration *

1) QRS complex The component phases should be identified in each lead At the same time abnormalities in contour of any phase of the QRS complex (namely notching or coarse slurring) should be noted and the time of occurrence after the onset of the QRS should be measured accurately One should then determine whether the deformity is coincident with the peak of a normal R wave in a nearby lead or whether it is an independent and abnormal phenomenon

a) Direction of the initial deflection of the QRS should be noted in each right ventricular lead and in each left ventricular lead *If a Q wave is found the following* procedure is recommended to determine its significance

(1) Analysis of each individual lead with an initial downstroke The manner of analysis depends upon whether or not a succeeding R wave is present

(a) Q wave succeeded by an R (occurring as a part of a diphasic qR or triphasic qRs and qRS) The interpretation depends not only upon the ventricle over which the pattern is recorded but also upon the following additional measurements

*The capital letters QRS are used as a general symbol of electrocardiographic recordings during ventricular activation and are not mistaken for an indication of 3 phases *of approximately equal amplitude* because of the rarity of such a finding

(1)) Time elapsing from onset to nadir of Q The measurement is made on a horizontal plane from the point on the upper margin of the baseline where the convexity of the Q wave begins to the apex of the Q wave

(2)) Amplitude of the Q wave (measured from bottom of the baseline to nadir as compared with the amplitude of the succeeding R (measured from top of baseline to peak)

(b) QS complex The interpretation depends not only on the ventricle over which the pattern is recorded but also upon the contour One should note whether the QS complex has smooth descending and ascending limbs or whether there is notching or coarse slurring representative of an r equivalent (i e embryonic r) on either limb

(2) Comparison of the initial deflections in the various right ventricular leads to determine whether the findings are normal or abnormal A diphasic qR and triphasic qrS complex in leads over the right ventricle are always abnormal on the other hand a QS may be found as a normal variant in leads near the right atrium

(3) Comparison of the Q waves in the various left ventricular leads as to duration and amplitude to determine whether the relationship is normal or abnormal A QS complex in a lead facing the epicardial surface of the left ventricle is always abnormal a qR complex may be normal or abnormal depending on (a) the duration of the Q and the ratio of Q to R amplitude in the individual lead (b) the relationships among the several leads

b) Relative amplitudes of the R and S deflections in the various right ventricular leads in the various left ventricular leads and in the former as contrasted with the latter The relative amplitude of the R in respect to the S is stressed rather than the absolute voltage of either because of the fact that extracardiac factors such as thickness and electrical conductivity of the intervening lung and thoracic cage markedly influence absolute voltage of any given deflection but do not modify relative amplitudes of 2 different phases of the QRS When the R wave is double peaked consisting of an R and R' component the measurements from the upper margin of the baseline to the top of the first peak (R) and from the onset of the second upstroke (or from the top of the isoelectric line if there is an intervening S) to the second peak (R') are taken when the S wave is doubled the measurement of the S' component is taken

(1) Findings in right ventricular leads

(a) Over-all findings Note whether the normal relationship (i e a relatively small r in comparison to S) prevails in all right ventricular leads or whether an abnormally large R in respect to S is present in one or more leads If an R' deflection is present note location of lead in which it is maximal and measure voltage to determine whether a normal variant or whether abnormal

(b) Comparison of patterns in the various right ventricular leads In comparing successive leads between right atrium and transitional zone note whether the expected progressive increase in the amplitude of the R wave at the expense of the S takes place or whether a paradoxical decrease or other abnormality is present

(2) Findings in left ventricular leads

(a) Over all findings Note whether the normal relationship (i e a relatively tall R and small or absent S) prevails in all leads or whether the R wave

is abnormally reduced in amplitude in respect to either the Q or S

(b) Comparison of patterns in the various left ventricular leads In comparing successive leads taken leftward from the transitional zone, note whether the expected progressive decrease and disappearance of the S wave takes place or whether the S wave persists into axillary and back leads

c) Duration of the ascending limb of the R wave and time of onset of the intrinsicoid deflection in a representative right ventricular lead as contrasted with corresponding measurements in a representative left ventricular lead In making the selection the lead expected to give maximal recording of the potentials developed in the activation of the one ventricle with minimal influence of forces from the opposite ventricle is chosen for measurement A lead over the tricuspid ring as judged by position of electrode and contour of the P wave is preferable for the right ventricle the lead with the largest R and no S wave is selected for the left ventricle If an S wave is present in all available leads back leads including V_9 and HV_9 should be taken in search for a lead without an S wave The duration of the ascending limb of the R wave is measured on a horizontal plane from its onset to peak the time of onset of the intrinsicoid deflection comprises the time elapsing between the onset of the QRS and the peak of the R (or R') deflection

(1) Duration of the ascending limb of the R wave When the R wave constitutes the initial deflection measurements are made from the beginning of the upward movement of the bottom of the baseline to the peak of the R (or to the peak of the R' instead if the latter is present) Such a measurement in a right ventricular lead will comprise the time required for activation of the septum plus the free wall of the right ventricle This measurement may not represent the actual time consumed since opposing negative potentials transmitted from the posterolateral wall of the left ventricle tend to foreshorten the R wave registered in right precordial leads This effect is exemplified by the exaggeration of the amplitude and duration of the R wave in right precordial leads after infarction of the posterolateral wall of the left ventricle In a left ventricular lead when septum is activated from right to left the time from beginning to peak of the R wave will represent the total interval for activation of the septum plus the free wall of the left ventricle When the R wave is preceded by a Q measurements are made from the nadir of the Q to the peak of the R (or R') This measurement provides an index of the time consumed in passage of the impulse from the endocardial to the epicardial surface of the subjacent outer wall

(2) Time of onset of the intrinsicoid downstroke is measured from the onset of the QRS to the peak of the last upstroke (R or R') wave This measurement will correspond with the duration of the ascending limb of the R if the initial deflection is an upstroke but will be longer if a Q wave is present The qR duration provides an index of the time elapsing between the onset of ventricular activation and the arrival of the impulse at the epicardial surface beneath the electrode

2) S-T complex The electrocardiographer should have knowledge of the age of the patient and of recent medication especially digitalis when evaluating the S-T complex Determination of the length of the Q-T interval in the tracing under study as compared to the average and upper limit of normal for sex and heart rate is essential to the interpretation of the S-T segment and T wave The following features should be evaluated in right ventricular leads and in left ventricular leads

a) S-T junction The position should be determined in millimeters above or below the T-P segment in a portion of the tracing where the T-P segment is at the

same horizontal level for 2 or more successive cycles and where no artefacts are present in the QRS T complex If the segment between the end of the P and onset of the QRS (P-R segment) is depressed below the T-P level allowance must be made for the amplitude of the causative T_p wave in judging the position of the S T junction It is customary to express differences in levels as if the T-P segment represented the isoelectric line and the S-T junction the degree of deviation from zero potential whereas differences in levels may actually be due to deviation of either or both from zero potential In the interpretation of displacement of the S-T junction the contour of the S-T segment and the amplitude and direction of the T wave must also be taken into consideration

b) S T segment It is often difficult or impossible to demarcate the end of the S-T segment from the beginning of the T wave and it is unnecessary to do so since the duration of the S-T segment is of minor clinical significance More important are the general contour and direction of the tracing between the S-T junction and the apparent beginning of the T wave The contour should be described in accordance with the curvature of the upper border of the tracing as concave convex or straight and the direction should be classed as either upward downward or horizontal

c) T waves Note should be taken of the following features

(1) Direction T waves are classed as upright flat inverted or diphasic The latter are subdivided into the +- variety (initial upright and final downward phases) or into the -+ type

(2) Amplitude is measured from the top of the isoelectric line to the summit of an upright T or from the bottom of the isoelectric line to the nadir of an inverted T

(3) Contour The shape of the summit should be noted and the gradient of the proximal and distal limbs compared In the presence of notching the possibility of a superimposed P wave or a partially fused U wave should be investigated Notching inherent in the T wave is of no significance when limited to the transitional zone and is rare in other leads

d) U waves Notation should be made of U waves that are of unusual amplitude or opposite to the direction of the T wave

2 [illegible] lations of the cardiac surface is applied (b) the position of [illegible] the direction of rotation on its transverse anteroposterior and longitudinal axes

[illegible] unipolar lead from each limb as compared with that in multiple precordial and/or esophageal leads The results of such comparisons indicate that the P and QRS T pattern in a given unipolar limb lead resembles that recorded in thoracic leads near the cardiac surface directly opposite the somatic attachment of the extremity and therefore serves as an index of the potential variations of this cardiac surface

Since the right arm faces towards the atria and great vessels the major deflection of ventricular origin in Lead aV_R is derived from the potential variations of the endocardium and cavities of the 2 ventricles as transmitted through the valvular orifices On the other hand the left leg faces towards the inferior surface of one or both ventricles and consequently Lead aV_F records the potential variations transmitted from the ventricular epicardium When the diaphragm is low to intermediate in position the potential variations of the left leg are governed largely by those of the epicardial

surface of the left ventricle as shown by the resemblance of the pattern in aV_F to that in chest leads over the left ventricle *when the diaphragm is high* the pattern in Lead aV_F resembles that in right ventricular leads *when moderately elevated* an equiphasic transitional complex of low voltage is recorded representing a cancellation of potentials of right and left ventricular origin

The findings in Lead aV_L tend to be opposite to those in aV_F and show even greater variation due to the fact that the potential variations of the left arm may be transmitted not only from the epicardial surface of either or both ventricles but also from the endocardial surface through the atrioventricular orifices The cardiac positions that facilitate transmission of potentials from the ventricular epicardium to the left arm tend to favor the registration of an upright P wave in aV_L in the presence of sinus rhythm whereas those positions that facilitate transmission of potentials from the ventricular endocardium to the left arm tend to favor the registration of an inverted to diphasic P wave in Lead aV_L

After identification of the surface of the heart having the predominant effect on the potential variations of each extremity the evaluation of the findings in the respective unipolar limb lead is based on whether or not they come within the expected normal range for the cardiac surface which they reflect For example if it is concluded that the potential variations of the left leg are transmitted principally from the epicardial surface of the left ventricle the criteria used for the evaluation of the findings in axillary and back leads over the left ventricle are applied in the interpretation of Lead aV_F Furthermore if it appears that the potential variations of the left arm are transmitted from the epicardial and not the endocardial surface of the left ventricle the same criteria may be applied

Lead aV_F furnishes a valuable index of the potential variations of the diaphragmatic surface of the heart (i e usually the apical half of one or both ventricles) It should be taken habitually both in the initial and repeat tracings because it provides information about a portion of the ventricles not adequately explored by the routine chest leads Lead aV_R provides direct evidence of injury to the endocardial surfaces of the ventricles and should be taken serially under such conditions Lead aV_L supplies little or no evidence not more reliably provided by chest leads

b Determination of the electrical position of the heart in the chest The direction of rotation on the transverse axis is judged from the pattern in aV_R and the direction of rotation on the anteroposterior and longitudinal axes from the comparative patterns in Leads aV_L and aV_F as will be brought out in the description of the normal findings (page 122)

3 Standard limb leads are still taken for sake of precedent but yield no information that *cannot be obtained better from the unipolar limb leads plus multiple chest leads* Therefore, the findings in standard leads will not be described If desired the findings may be determined algebraically from the recordings in the two constituent unipolar limb leads

C THE ORIGIN AND FORM OF THE NORMAL AND ABNORMAL QRS T COMPLEX

Variations in electrical potential associated with activation and repolarization of an isolated cell Variations in electrical potential are not unique to cardiac muscle but have been demonstrated during the activation of other types of excitable tissue from plant as well as animal sources The physiologic basis for the QRS and T deflections of the electrocardiogram may be better understood through a preliminary consideration of the electrical phenomena recorded during the activation of a single cell

Successful recordings have been obtained from isolated cells of the alga Nitella and the giant axon of the squid which are several centimeters in length and approximately 5 mm in diameter By insertion of an electrode into the cell and pairing with another electrode on the surface measurements of the impedance (electrical resistance) of the cell membrane and the voltage across it have been made The membrane of the resting cell possesses a high impedance the potential of the outer surface exceeding that of the interior by 50 100 mv The high impedance of the cell membrane is represented by the heavy line of Fig 2 a and the potential distribution by the plus and minus signs The intact resting cell is in a polarized state

Although any point on the external surface of the intact resting cell is positive in reference to the interior it is at the same potential as any other point on the exterior as shown by the fact that no deflection will be recorded when a galvanometer is connected to electrodes at Y and Z or at any other points on the external surface The illustrations of Fig 2 represent tracings that would be obtained by the separate recording of the potential variations at the two ends of the cell through the use of two galvanometers The first galvanometer is connected to point Y adjacent to the left end of the cell and to a distant electrode far enough away so that its potential variations are negligible the second galvanometer is connected to point Z at the right end of the cell and to a similar remote electrode Connections are made so that an upright deflection will be recorded when the potential at the adjacent surface electrode is relatively positive and a downward deflection when it is negative

The application of an appropriate stimulus to one end [illegible] cell (at Y in Fig 2 b) causes a [illegible] as represented diagrammatic [illegible] by a thin line and produces a short circuit Posit[illegible] from the stimulated end disappear through the altered membrane into the cell to expose the negativity of the interior at point Y as represented by the arrows in Fig 2 b The potential at the point of excitation suddenly reverses becoming negative in respect to the positive potential of the portion still in the resting state as indicated by the downward deflection recorded through the electrode at point Y and the upward deflection at point Z An electrical bipole is thus set up at the boundary zone between the portion of the cell which has just become active and the portion still in the resting state much as if a small battery with negative and positive poles straddling this boundary zone had been activated

The spread of the exciting impulse through the cell is accompanied by a sudden fall of impedance of contiguous portions of the membrane through which current continues to flow from the outer surface into the interior as indicated in Fig 2 c d The electrical bipole marking the boundary between the active and resting portions moves progressively across the cell Throughout the spread of the excitation wave the potential of the end of the cell to which the stimulus had been applied becomes more negative as indicated by the progressive downward deflection recorded through the electrode at Y, whereas the opposite end continues positive as indicated by the steadily rising deflection recorded through the electrode at Z The fact that the progress of the excitation wave is slow enough to be photographed on moving film is in keeping with the fact that a chemical impulse is the basis for the changes of potential The latter are transmitted instantaneously and registered in accordance with the laws of electrical conduction

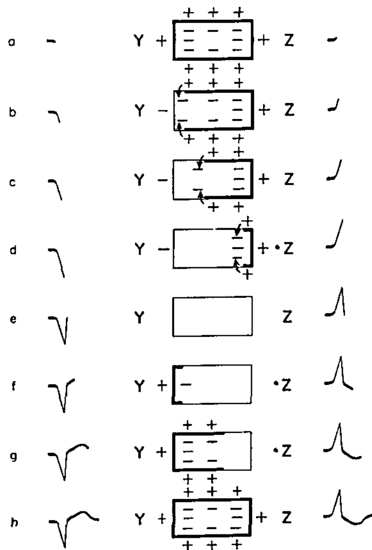


Figure 2

Upon arrival of the excitation wave at the opposite end the last portion of the cell changes from resting to active state the electrical bipole is extinguished and current flow ceases The potential at both ends suddenly reverts to zero as indicated by an abrupt vertical return of the tracings at Y and Z to the starting level in Fig 2 e Thus an upright deflection or R wave is completed from the recordings at Z whereas an equal but oppositely directed deflection or QS wave is completed from the recordings at Y These deflections are thus a manifestation of activation or depolarization of the cell

If the depolarized state depicted in Fig 2 e, persisted for an appreciable time one would expect that the tracing would run horizontally on the isoelectric line of zero potential after the completion of the QRS deflection Recordings through the electrode at Y show that the ascending limb of the QS complex continues above the isoelectric line to undergo an abrupt transition (the S-T junction) into a more gently sloping upward deflection (the S-T segment and T wave) illustrated in Fig 2 f This indicates that the electrode at Y has become electropositive by the time activation is completed In the recordings through the electrode at Z the descending limb of the R wave continues below the isoelectric line where it undergoes an abrupt transition into a more gradually sloping downward deflection indicating that the electrode at Z has become negative by the time activation is completed The reappearance of a positive potential at Y and a negative potential at Z is accompanied by a measurable rise in the impedance of the cell membrane at the point of application of the original stimulus The return of high membranal impedance takes place much more gradually than the precipitous fall during activation but pursues the same course developing first in the portion of the cell adjacent to Y and progressing to the opposite end as indicated in Fig 2 f g h The return of high membranal impedance is accompanied by the reappearance of a potential difference across the cell membrane and constitutes the process of repolarization

The representation of repolarization in a series of three diagrams and activation in a series of four was made for convenience of description and is in no way indicative of the relative amounts of time consumed in the two processes The temporal relationships are brought out through the recordings the steep slopes of the QRS its thin image and the brief interval from beginning to end of activation contrasting sharply with the gradual slope of the T its thick image and the relatively long interval from the S-T junction to the end of the T wave * During the first portion of repolarization a gently sloping upstroke is recorded at Y indicating gradually increasing positivity of the surface of the cell facing point Y Coincidentally a gently sloping downstroke is recorded at Z indicating that the gradually increasing negativity of the interior is being referred through the as yet depolarized cell membrane facing point Z During the latter part of repolarization the tracing at Z shows a gradual rise to the isoelectric line indicating a progressive resolution of the former negativity by return of positive potential to the surface adjacent to point Z Meanwhile the tracing at Y returns to the isoelectric line due to balancing of the positive potential at Y by the developing positive potential at Z thereby completing repolarization It is noteworthy that the T wave recorded from a single cell is opposite to the main deflection of the QRS After the completion of the T wave the cell has returned to the resting or polarized state

<u>The origin and form of the QRS T complex as recorded in direct leads from the mammalian ventricle</u> During every cardiac cycle each of the functioning muscle cells in the myocardial syncytium undergoes depolarization and subsequent repolarization analogous to that which occurs in the solitary cell of Nitella and the giant axon of the squid Modern concepts of ventricular activation and repolarization advanced by Lewis and elaborated by many others are illustrated by a series of diagrams in Figs 3 and 4

In Fig 3 only four of the large number of cells that make up the thickness of the ventricular wall are depicted The polarized state characterized by a high impedance of the cell membrane with a positive potential on the external surface and a negative potential in the interior is indicated in these diagrams by employing a heavy line for the cell membrane The depolarized state characterized by loss of membranal impedance and disappearance of the potential difference across it is indicated by the use of a light line for the cell membrane Cells undergoing activation in Fig 3 b c d and those undergoing repolarization in Fig 3 f g are outlined in part by a light and in part by a heavy line and the resultant electrical bipole is indicated by a + and − sign

The series of diagrams in Fig 3 reconstruct schematically the tracings obtainable by separate recording of the potential variations of the epicardial surface and cavity of the mammalian left

* The width of the photographic image is inversely proportional to the speed of string movement

ventricle through the use of two galvanometers after the manner of Wilson and associates One terminal of the galvanometer is attached to an exploring electrode in contact with the heart whereas the other terminal is made almost completely indifferent by connection to the two forelegs and left hind leg through the Wilson central terminal The exploring electrode connected with the first galvanometer consists of a sharply pointed insulated wire which is thrust through the ventricular wall so that the bare tip projects into the ventricular cavity at point Y and registers potential variations of the cavity the remainder of the wire being adequately insulated from currents in or exterior to the ventricular wall The exploring electrode of the second galvanometer is applied to the visceral pericardium at point Z in the diagram thereby registering the potential variations of the epicardial surface of the small segment of ventricular wall to which it is attached The diagrams represent the tracings obtained with connections made in such a way that a positive potential in the exploring electrode will register as an upright deflection whereas a negative potential will register as a downward deflection The series of diagrams depicts a single cycle broken up into the following stages ventricular diastole Fig 3 a spread of the activating impulse from endocardium to epicardium Fig 3 b c d extinction of the activating impulse at the epicardial surface Fig 3 e repolarization Fig 3 f g h

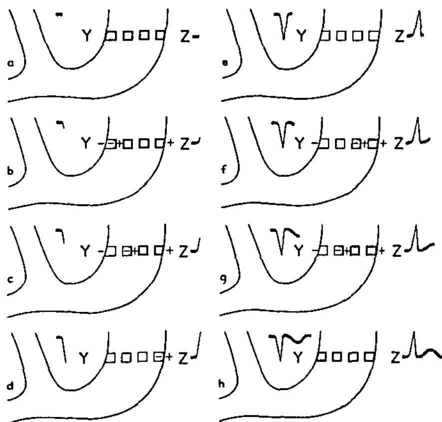


Figure 3

In Fig 3 a representing ventricular diastole all of the cells are electrically polarized comparable to the single cell of Fig 2 a As long as the entire ventricular wall is in the resting state no potential difference will exist between the endocardial and epicardial surface and no deflection will be recorded by either galvanometer The next event is depicted in a series of 3 diagrams Fig 3 b c d The progressive descent of the tracing at Y signifies increasing negativity of the left ventricular cavity the simultaneous upstroke at Z denotes reciprocally developing positivity of the epicardial surface The diagrammatic evolution (Fig 3 b c d) begins with the arrival of the impulse at point Y via the Purkinje network it reflects the centrifugal spread of the impulse towards the epicardium and successive activation of each responsive cell in its pathway As the impulse reaches a given cell it produces a sudden decrease in the impedance

of the cell membrane accompanied by an abrupt drop in electromotive force across this membrane The cell undergoing excitation becomes negative in respect to the resting cells superficial to it and current flows from points just ahead of the impulse into the cell just behind it The negative potential of the cell undergoing activation is transmitted backward through the intervening deeper layer of myocardium into the cavity as indicated by the downward deflection registered through the electrode at Y and reproduced in Fig 3 b c d The relatively positive potential of the cell just ahead of the advancing impulse is transmitted through the intervening resting muscle to the epicardium as indicated by the upward deflection simultaneously registered through the electrode at Z

Transmission of positive potential to the epicardial and cutaneous surfaces facing the advancing impulse occurs because the intervening resting myocardium and soft tissues serve as electrical conductors Negative potentials are simultaneously transmitted through activated myocardium to the endocardial surface thence through the blood in ventricular and auricular cavities the atrial wall and soft tissues to the opposite cutaneous surface From the standpoint of electrocardiographic methods of recording transmission of potential changes to any point in the body may be regarded as instantaneous since it takes place in accordance with the laws of electrical conduction On the other hand passage of the impulse through the auricles and ventricles is slow enough to permit photographic registration and timing on film moving as slowly as 1 mm per 04 second

When recordings are made through an electrode applied to the epicardium at point Z an upstroke presumably makes its appearance the moment the impulse reaches and begins to activate the underlying subendocardial layer (Fig 3 b) continues to rise as long as the impulse is advancing through responsive muscle (Fig 3 c) and attains its peak upon arrival of the impulse at the epicardial surface (Fig 3 d) When the most superficial muscle lying just beneath the epicardium has been activated the potential difference across the ventricular wall is abruptly extinguished as depicted in Fig 3 e The potential at point Z suddenly drops to zero as manifested by an almost perpendicular downstroke known as the intrinsic deflection The time required for the impulse to pass through the ventricular wall is thus represented by the interval elapsing between the onset and the peak of the R wave and depends upon the thickness of the ventricle and the integrity of the muscle composing its wall

Coincident with the registration of the upstroke of the R wave through epicardial Lead Z a reciprocal downstroke is recorded through cavity Lead Y (Fig 3 b c d) After complete activation of the ventricular wall there is an abrupt return of cavity potentials to zero mainifested in the tracing at Y by an abrupt ascent to the isoelectric line (Fig 3 e)

After a brief but variable interval at the isoelectric level corresponding to the interim between completion of activation and onset of repolarization the tracing at Z normally describes a gentle upward slope whereas that at Y shows a reciprocal downslope as illustrated in Fig 2 f and g The deflection in process of formation is the T wave and is a manifestation of repolarization

The fact that the normal T wave recorded by an electrode in contact with the epicardium or facing this surface is upright whereas that recorded by an exploring electrode within the ventricular cavity is inverted would signify that the epicardial surface is relatively positive and the endocardial surface relatively negative during the process of repolarization Thus the T waves respectively recorded from surfaces of origin and termination of the activating impulse are the opposite of T waves recorded from corresponding surfaces of the single muscle cell depicted in Fig 2 f g h This would indicate that repolarization of the normal mammalian ventricular wall begins subepicardially and progresses centripetally towards the endocardium as depicted in Fig 3 f g h In other words the subepicardial muscle passes out of the excited state ahead of the deeper muscle and thus becomes positive relative to the subendocardial layer The predominant effect of the subepicardial layer of muscle on the configuration of the T wave in leads facing the ventricular surface is exemplified by the striking changes in the T wave in pericarditis and in other lesions limited to the subepicardial layer of myocardium On the other hand the subendocardial layer has the predominant effect on T waves recorded through cavity leads

In Fig 3 attention was focussed on the effect of electromotive forces derived from a small segment of the ventricular wall upon the potential variations of a direct lead from its epicardial surface no consideration was given to the possible influence of electromotive forces generated elsewhere in the heart The tracing obtained through a direct epicardial lead actually represents an algebraic summation of potentials transmitted to the electrode from all portions of the heart undergoing activation or repolarization however the influence of excited muscle in any given portion of the heart upon the potential variations of an epicardial electrode is inversely proportional to the cube of the intervening distance For this reason, the portion of the tracing recorded through an epicardial electrode during activation or repolarization of the underlying segment of muscle will represent largely but not quite exclusively the potential differences developed across this segment of muscle

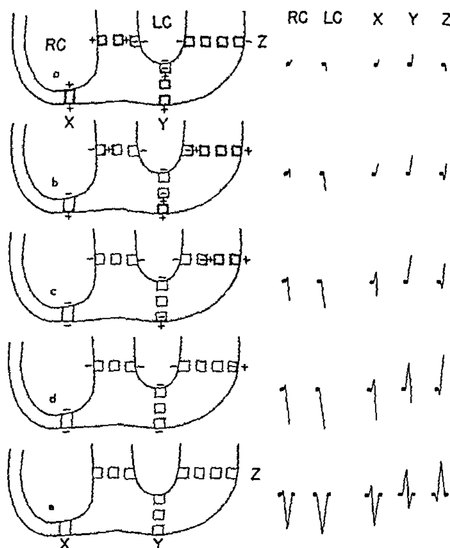


Figure 4

The amplitude of the R and T waves in a *direct epicardial* lead thus serves as an index of the voltage developed in the underlying wall during activation and repolarization respectively The effect of electromotive forces released simultaneously in the opposite wall of the heart on the R and T waves in direct epicardial leads is relatively small because of the cube root decrement in *potential* with increasing distance but nevertheless is indirectly demonstrable by the exaggeration of the R and erect T waves in leads over the anterior wall following infarction of the posterior wall Furthermore the transmissibility of potentials of *remote origin through* the excited as well as the resting ventricular wall is exemplified in complete auriculoventricular block by the registration in ventricular leads of recognizable P waves superimposed on the QRS and T waves

as well as on the isoelectric line The chief influence of activation of the opposite wall or more remote portions of the heart on a tracing through a direct epicardial lead occurs during the interval prior to the arrival of the impulse in the subendocardial layer of the underlying segment and during the period following extinction of the impulse at the epicardial surface If activation of the opposite wall is in progress during either period negative potentials referred to the cavity may be transmitted to the epicardial electrode and recorded as a downstroke reaching below the isoelectric line

The QRS complex recorded in direct or in precordial leads is usually not monophasic as illustrated in Fig 3 It is most commonly diphasic consisting either of an RS or a qR deflection not infrequently it is triphasic exhibiting a q an R and an S wave In Fig 4 an attempt is made to illustrate diagrammatically the mode of origin of the normal q and S waves This figure reconstructs tracings that would be obtained by simultaneous leads from five separate points namely LC the cavity of the left ventricle RC, the cavity of the right ventricle X the epicardial surface of the anterior wall of the right ventricle Y the epicardial surface of the anteroseptal wall of the left ventricle Z the epicardial surface of the lateral wall of the left ventricle The normal difference in thickness of the right and left ventricular walls has been depicted by representing the former by a single cell the latter by three to four cells The same symbols have been employed as in Fig 3 Fig 4 a represents the portion of the tracing recorded during the first 01 after the arrival of the impulse together with the probable state of ventricular activation The next three figures represent the progress during successive intervals of approximately 01 sec each whereas the last figure covers the final portion of ventricular activation *

Reference to Fig 4 a reveals that the left ventricular cavity (LC) is initially negative the right (RC) positive indicating that the impulse normally reaches and begins to activate the left side of the septum slightly ahead of the right During this period the lead from the epicardial surface of the right ventricle (X) shows an upstroke synchronous with that recorded in Lead RC The beginning of the R wave at X is thus produced by transmission of positive right ventricular cavity potentials through the as yet unactivated outer wall of the right ventricle A lead from the anteroseptal surface of the left ventricle (Y) also shows an initial upstroke Since the left ventricular cavity is coincidentally negative the positivity at the surface denotes early onset of activation of the subendocardial layer of the anteroseptal wall of the left ventricle as indicated in Fig 4 a On the other hand an initial downstroke or q wave is recorded in the lead from the epicardial surface of the lateral wall (Z) paralleling that in Lead LC and resulting from transmission of cavity potentials through the as yet unactivated lateral wall The normal q wave recorded in leads from the lateral and posterior walls of the left ventricle is an expression of the physiologic interval of 01 to 02 sec between onset of septal activation and the arrival of the impulse in the lateral and posterior walls and is attributable to the finding that the impulse requires 01 sec to spread approximately 40 mm through the Purkinje network

During the next 01 sec (Fig 4 b) Lead RC shows replacement of the initial R wave by a downstroke whereas Lead X from the surface of the right ventricle shows further increase in the R wave These findings are attributable to activation of the right side of the septum in a right-to-left vector together with activation of the free wall of the right ventricle from endocardium to epicardium The ascending limb of the R wave at X is thus initiated by early left-to-right septal activation and is continued by spread of the impulse from the endocardial to epicardial surface of the right ventricle The attainment of a peak by the end of 02 sec signals the arrival of the impulse at the epicardial surface of the right ventricle Meanwhile there is a progressive rise in the upstroke at Y referable to advance of the impulse through the anterior left ventricular wall The direction of the tracing at Z is reversed the replacement of the q by an upstroke marking the onset of activation of the subendocardial layer of the lateral wall

* For purposes of description intervals have been arbitrarily selected to approximate those found in the human rather than in the relatively small canine heart

By the end of 03 sec (Fig 4 c) the attainment of the peak of the R wave at Y is an expression of the arrival of the impulse at the epicardial surface of the anterior wall of the left ventricle but *the continued ascent at Z is referable* to continued progress through the lateral wall The negative potentials simultaneously referred to the cavity during activation of the free wall of the left ventricle are *manifested by a progressive downstroke in the LC lead* A parallel downstroke now appears in leads from the cavity and surface of the right ventricle due to transmission of left ventricular cavity potentials through the depolarized *septum and right ventricle to points RC and X* Thus the S wave in these right-sided leads is a manifestation of the same forces that are responsible for the R wave in leads from the epicardial surface of the left ventricle

After approximately 04 sec (Fig 4 d) the attainment of the peak of the R wave at Z indicates that *the impulse has reached the epicardial surface of the lateral wall* Meanwhile the intrinsic deflection has been inscribed at Y and has continued downward below the isoelectric line as an S wave due to transmission *of negative cavity potentials through the depolarized anterior left ventricular wall* Further increase in the S waves at X and RC is evident paralleling the downstroke at LC

Fig 4 e represents the abrupt return of cavity and surface potentials to zero following completion of ventricular activation *Thus a QS complex is recorded from LC* signifying that the left ventricular cavity is negative throughout ventricular activation an rS is recorded from RC and X indicating initial but *brief positivity followed by prolonged negativity The initial R at Y shows* that activation of the anteroseptal wall of the left ventricle began early, but the succeeding S indicates completion ahead of other portions of the left ventricle *The qR complex without succeeding* S at Z is evidence of relatively late onset and completion of excitation of the lateral wall Since the T wave registered over the anterior and lateral walls of the right and left ventricles is normally upright, the electrical events associated with its inscription are presumably similar to those illustrated in Fig 3 f g h The T wave in right as well as in left ventricular cavity leads is normally inverted reflecting the slower repolarization of the endocardial than the epicardial surfaces

Comparison of direct and precordial leads The discussion thus far has been based upon diagrams of records obtainable by direct leads from the cavity and from various points on the epicardial surface of the mammalian ventricle Such leads are not feasible in man except in the occasional patient undergoing cardiac surgery or catheterization The closest approach that can be achieved routinely in the human is the semidirect lead in which the exploring electrode is applied to various points on the thorax or inserted at different levels in the esophagus whereas the other electrode is made as indifferent as possible by connection *to the two arms and left leg* via the central terminal Under these circumstances the potential variations of the indifferent electrode *are reduced to a minimum* and probably never exceed 3 mv

A fairly close correspondence has been demonstrated between tracings obtained by direct leads from the epicardium in dogs and tracings recorded *simultaneously by semidirect leads from* the overlying precordium of the same animal The observed differences could be anticipated from the nature of the leads The amplitude *of the deflections in direct leads is much greater* than in precordial leads in keeping with the demonstration that voltage decreases in proportion to the cube *of the distance from* the source of the electromotive forces

The shape of the deflection in the precordial as compared to the direct leads will depend upon whether or not the segment of ventricular wall which dominates the potential variations of the precordial lead is uniform in electrophysical activity The reason for this becomes apparent when one compares the areas of the segments of ventricular wall which have the dominant influence on the R and T wave as recorded by epicardial as compared to precordial leads The electrode employed for epicardial leads generally consists of a thread moistened with saline and the R and T waves recorded thereby represent almost exclusively the potentials derived from *the small core* of wall to which the electrode is applied the electrodes used clinically on the thorax are metal disks 3 cm in diameter Furthermore *electrodes applied to the skin or esophageal mucous*

membranes are separated from the epicardium by soft tissue and thus subtend a still larger area of the cardiac wall

If the segment of wall which dominates the potential variations of the precordial lead is normal or uniformly abnormal in electrophysical activity the shape of the tracing will resemble that recorded from any point within the circumference of the zone subtended by the precordial lead As would be expected the ascending limb of the R wave in a precordial lead is not as steep as in any underlying direct lead because the former is a composite derived from a relatively large segment of the myocardium in various parts of which there are slight differences in time of onset and extinction of activation For the same reason the downstroke following the peak of the R wave is practically perpendicular in a direct lead but less abrupt in the corresponding precordial lead In recognition of these differences the descent of the R wave is referred to as the intrinsic deflection in a direct lead and the intrinsicoid deflection in a precordial lead

If there is a small circumscribed lesion within the zone that has the predominant influence on the precordial lead various direct leads from points on the epicardium within this zone may exhibit striking differences depending upon whether they overlie normal or abnormal myocardium The precordial electrode will record a mixture of effects from multiple direct leads and will yield a tracing resembling that in the majority of subjacent epicardial leads

Although the segment of ventricular wall which dominates the potential variations of a precordial lead is much larger than the segment which dominates the potential variations of a direct epicardial lead it nevertheless comprises only a fraction of the cardiac surface as exemplified by the fact that slight changes in the position of the exploring electrode on the thorax result in distinct changes in configuration of the tracing Thus multiple precordial leads studied in reference to one another will provide information sufficient for the detection and approximate localization of lesions of the anterolateral wall of the ventricles such as myocardial infarction whereas multiple esophageal and back leads will furnish analogous data for the diagnosis of infarction and other lesions of the posterior wall

To cover the anterolateral aspect of the heart Wilson recommended a minimum of six precordial leads namely V_1 through V_6 (F g 5) The electrode in positions V_1 and V_2 is in the fourth interspace at the right and left sternal borders respectively in these positions it generally faces towards the right ventricle or auricle and records a QRS dominated by the potential variations of the right side of the septum and epicardial surface of the right ventricle Since the electrode in the V_1 position is often not far enough to the right to advantageously register the potentials derived from activation of the tricuspid ring at least one additional lead from the right precordium is recommended either V_{3R} or V_{4R} In the V_3 and V_4 positions the electrode may be over the anterior wall of the right or left ventricle or may straddle the anterior margin of the septum depending upon the position and size of the heart and thus records a greater admixture of right and left ventricular effects than precordial leads farther to the right or left Positions V_5 and V_6 are located in the anterior axillary and mid axillary lines respectively at the same horizontal level as V_4 and thus normally face towards the anterolateral surface of the apex of the left ventricle Accordingly the QRS T complex in Leads V_5 and V_6 represents almost exclusively the potential variations of the distal portion of the left side of the septum and the epicardial surface of the anterolateral wall of the apex of the left ventricle To cover the posterolateral and posterior aspects of the ventricle requires the addition of Leads V_7 and V_8 taken in the posterior axillary and scapular lines respectively at the same horizontal level as V_6

As illustrated in F g 5 positions V_4 V_5 and V_6 face towards the [illegible] the left ventricle To cover the base of the heart leads taken [illegible]
are reco[illegible] terspace and sternum
the vic[illegible] HV_1 and HV_2 are in
domin[illegible] [illegible]ow tract of the right ventricle and may show
[illegible]st areas in the event of hypertrophy HV_3 HV_4 and sometimes even HV_5 are transitional and reflect the potential variations of the basal portions of the right or left ventricle depending upon cardiac position and size The QRS-T complex in Leads HV_5 HV_6 HV_7 and HV_8 is derived chiefly from activation of the proximal portion of the left side of the septum

and the anterolateral or posterolateral aspects of the base of the left ventricle Unipolar limb leads taken with an exploring electrode on either the right arm the left arm, or left leg, subtend a much wider surface area than precordial leads and thus are less definitive in localization The increased time and expense of registration of multiple precordial leads is fully justified by the fact that they frequently reveal abnormalities that cannot be detected in the limb leads

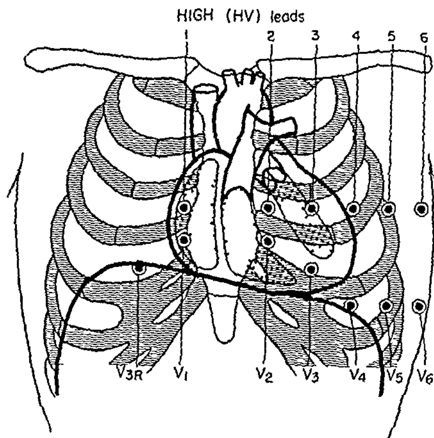


Figure 5

The form of the QRS complex as recorded in precordial leads of the normal adult In this subject the QRS deflection was 08 sec in duration and was normal in contour in all leads The precordial electrocardiogram is reproduced in Fig 6 and is analyzed through a set of four diagrams In the first drawing (Fig 6 a) that portion of the QRS which was registered in each lead during the first 01 sec after the onset of the complex is accurately reconstructed and is accompanied by a schematic reproduction of the probable state of ventricular activation at the end of the first 01 sec The second, third and fourth drawings carry on in a similar fashion respectively reproducing the portion of the QRS completed by the end of 02 04 and 06 sec together with the probable status of ventricular activation at the end of each period Since the precordial leads in this and in subsequent figures were taken consecutively rather than simultaneously this analysis is subject to error in the event that the beginning or end of the QRS complex happened to be isoelectric

The grid on which the electrocardiographic drawings were made was two and one-half times the square millimeter markings of the electrocardiogram taken at the customary camera speed This was done in order to separate the upstroke and downstroke of the QRS complex sufficiently so that each could be clearly identified in the drawing The time interval as measured on a horizontal plane between any two vertical lines is 04 sec and that between each fifth heavy line is 20 sec thus corresponding with the electrocardiographic tracing The amplitude of the tracing as measured in millimeters in the drawing is the same as in the original electrocardiogram and thus the amplitude is not subjected to the magnification (× 2 5) employed for the time intervals

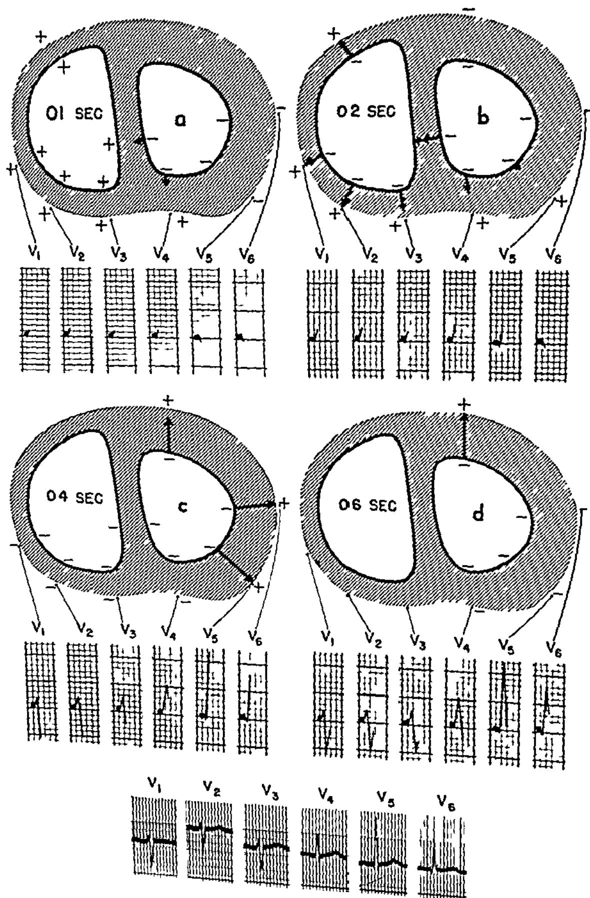


Figure 6

In the cross sectional diagrams the portion of the ventricle over which the electrode was presumably centered is indicated by a dot for each of the six leads Since the electrode was placed on the precordium at some distance from the epicardium it subtended a much larger ventricular area than is represented in the diagram The sign adjacent to each dot was determined from the direction that the string was taking up to the moment depicted in the diagram Thus the registration of an upstroke in a precordial lead indicates that the electrode was becoming increasingly positive and is represented by a plus sign at the corresponding epicardial dot the registration of a downstroke signifies that the potential of the exploring electrode is changing in the opposite direction and is represented by a minus sign The sign in the cavity denotes the presumed potential of the endocardial surface at the same moment

The course of the impulse through the segment of wall subjacent to each of the six precordial positions is represented by arrows The beginning of the upstroke in a given lead is taken as the reference point for the start of the impulse through the underlying subendocardial layer whereas the peak of the upstroke marks the arrival of the impulse at the epicardial surface The arrowhead represents the approximate position of the activating impulse at the moment depicted in the diagram as estimated from the portion of the ascending limb of the R wave completed at that moment When the phase illustrated in the diagram antedates the arrival of the impulse or follows the completion of its passage through a given segment of myocardium the arrow is omitted Under these circumstances potentials created by activation of other portions of the ventricle and referred to the cavity are transmitted through inert segments to dominate the exploring electrode as indicated in the figures by parallelism in the potentials of the endocardial and epicardial surfaces

Preliminary reference to the original electrocardiogram (Fig 6 e) reveals that the QRS complexes at V_2 and V_3 resemble closely that recorded at V_1 whereas the QRS complexes at V_4 and V_5 are much like that at V_6 The striking contrast in the form of the QRS complex in V_3 and V_4 indicates that the electrode in its shift from one position to the next has crossed the interventricular septum Thus in this case the electrode at positions V_2 and V_3 like that at V_1 is dominated by the potential variations of the epicardial surface of the right ventricle whereas the electrode at positions V_4 and V_5 like that at V_6 is dominated by the potential variations of the epicardial surface of the left ventricle

The initial upstroke in right ventricular Leads V_1 and V_2 indicates early positivity of the epicardial surface of the right ventricle Since an early R wave has been demonstrated in leads from the right ventricular cavity of dogs and human subjects the endocardial surface of the right ventricle is electropositive initially as illustrated in Fig 6 a The R wave in leads from the right ventricular cavity and the first portion of that in leads from the right precordium is derived from electromotive forces originating in the septum and is due to earlier onset of activation of the left side of the septum as illustrated in Fig 6 a and/or to greater magnitude of the forces developed through activation of the left than the right half of the septum as indicated by the disproportion between the septal arrows in Fig 6 b Passage of the impulse through the free wall of the right ventricle as represented in Fig 6 b contributes to the R wave in leads from the right precordium The attainment of the peak of the R wave in Leads V_1 and V_2 by the end of 02 sec and the onset of the intrinsicoid deflection immediately thereafter indicate early extinction of the positive potentials referred to the right during septal activation and early arrival of the impulse at the epicardial surface of the right ventricle as depicted in Fig 6 b The brief time required for the impulse to traverse the free wall of the right ventricle is attributable to the thinness of this structure in normal persons

The general resemblance of the tracing at position V_3 to that at V_1 and V_2 indicates that V_3 in this case is likewise dominated by the potential variations of the right side of the heart The greater amplitude of the R wave at V_3 than at V_2 and V_1 together with the later attainment of the peak, is attributable to greater admixture of positive potentials derived from activation of the nearby anteroseptal wall of the left ventricle

The initial upstroke in Lead V_4 taken from the vicinity of the anteroapical wall of the left ventricle is a manifestation of the early arrival of the impulse at the left apex as illustrated in Fig 6 a and as demonstrated in the exposed human heart The continuing ascent of the R wave in V_4 after a period of 02 sec signifies that the impulse is still in progress through the anteroapical wall of the left ventricle The significantly longer time required for activation of the anterior wall of the left than the right ventricle is referable to the normal difference in thickness

The fact that the initial movement of the string is upward at V_4 and downward at V_5 and V_6 indicates that the impulse has reached and begun to activate the anteroapical portion of the left ventricle subjacent to position V_4 prior to its arrival in the anterolateral and lateral walls facing positions V_5 and V_6 The antecedent onset of activation of the septum and anteroapical wall of the left ventricle causes increasing negativity of the ventricular cavity which is transmitted through the as yet unactivated lateral wall producing q waves at V_5 and V_6, as illustrated in Fig 6 a The downward movement or q wave at V_5 lasts for 01 sec when it is suddenly replaced by an upward movement or R wave marking the onset of activation of the subendocardial muscle of the anterolateral aspect of the left ventricle as represented by the arrowhead in Fig 6 b Activation of the lateral wall beneath V_6 begins somewhat later the downward movement of the string continuing for a total of 02 sec The fact that the q waves in V_5 and V_6 last but 01 and 02 sec respectively and are succeeded by R waves whose amplitude is at least four times as great indicates that these q waves are within normal limits Both their duration and magnitude are explainable by the normal difference in onset of activation of the septal and lateral walls of the left ventricle

An upward movement of the string begins in Lead V_6 immediately after the expiration of the 02 sec interval and marks the arrival of the impulse in the subendocardial layer of the lateral wall The activation of the lateral wall of the left ventricle proceeds rapidly thereafter and the peak of the R wave heralding the arrival of the impulse at the epicardial surface is attained by the end of 04 sec (Fig 6 c) Meanwhile the peak of the R wave at V_4 has been passed and the intrinsicoid downstroke is in progress at the end of 04 sec During the interval between 02 and 03 sec the upstroke of the R wave in right ventricular Leads V_1 V_2, V_3 has given way to an intrinsicoid movement which reaches the isoelectric line by the end of 03 sec This downstroke does not stop at the isoelectric line but continues below as an S wave due to the fa[illegible] that the left ventricular cavity potentials which a[illegible] activation of the lateral and posterior walls of t[illegible] [illegible]mpletely depolarized septum and right ventricula[illegible] pos[illegible] V_1, V_2 and V_3 By the end of 06 sec (Fig 6 d) the entire outer wall of both ventricles has been activated as shown by the fact that the string does not digress further from the isoelectric line in any lead During the interval from 06 to 08 sec the potentials of both the cavity and epicardial surfaces rapidly return to zero

The progressive increase in the amplitude of the R wave and in the length of the interval from the onset to the peak of the R wave as the electrode is moved from position V_1 to V_5 is a normal finding and merely reflects the normally increasing thickness of myocardium beneath the exploring electrode

The interval from onset to peak of the R wave in Leads V_1 and V_2 provides an index of the time elapsing between the onset of septal activation and the arrival of the impulse at the epicardial surface of the right ventricle and the measurement of 02 sec represents a typical normal finding The interval from the onset of the QRS complex to the peak of the R wave in Leads V_5 and V_6 provides an index of the time elapsing between the onset of septal activation and the arrival of the impulse at the epicardial surface of the anterolateral wall of the left ventricle and the measurement of 04 sec is below the normal limit of 05 sec The interval from onset to the peak of the R wave in V_5 and V_6 furnishes an index of the time required for the impulse to pass from the endocardial to the epicardial surface of the anterolateral wall of the left ventricle and the maximal measurement of 03 sec is below the normal limit of 04 sec

The fact that the S wave is deepest at V_1 and V_2 and progressively diminishes as the electrode is moved farther to the left is an aftermath of the normal differences in time of completion of

activation The S wave is deepest over portions of the ventricles where activation is completed earliest, because of the longer time interval available for the transmission of negative cavity potentials to the overlying precordial electrode The absence of an S wave from Lead V_6 indicates that the lateral wall was one of the last portions of the ventricles to become depolarized the cavity potential having returned to zero by the time the intrinsicoid deflection was completed in this lead

The form of the QRS complex as recorded in esophageal leads of the normal adult The form of the tracing in esophageal leads depends upon the position of the electrode in reference to the left atrium and the two ventricles This is exemplified by Fig 7 selected esophageal thoracic and limb leads obtained upon a normal subject are reproduced in Fig 7 e and correlated with anatomic diagrams in Fig 7, a b c d

A rough index of the position of the esophageal electrode may be obtained from the length of catheter passed through the nares, but a more precise estimate of its relation to the left atrium is based upon the contour of the P wave, provided that the pacemaker is in the sinus node The presence of a precipitous intrinsicoid downstroke in the P wave as evident in Leads $E_{35,40}$ of Fig 7, e indicates that the electrode is behind the left atrium When the electrode is near the auriculoventricular groove the intrinsicoid downstroke is almost entirely above the isoelectric line as in Lead E_{40} When the electrode is near the upper margin of the left atrium the major portion of the intrinsicoid deflection extends below the isoelectric line

The intrinsicoid auricular deflection is absent when the electrode is well above or below the atrium and the P wave exhibits a more gentle slope and a more rounded contour In the presence of normal sinus rhythm the P wave registered from points in the esophagus above the left atrium is inverted whereas that recorded from points below the atrium and behind or below the ventricle is upright An upright P wave with gently sloping limbs signifies that the electrode is either in the lower esophagus opposite the posterior wall of the ventricle or in the stomach facing the inferior diaphragmatic surface of the ventricles The contour of the P wave in Lead E_{55} of Fig 7 indicates that the electrode was below the left atrium and the short stature of the patient suggests that it was lodged in the stomach Under these circumstances the QRS-T pattern may be considered representative of the potential variations of the epicardial surface of the posterior diaphragmatic ventricular wall The decision as to whether the predominant influence came from the posterior inferior wall of the left or the right ventricle is based upon a comparison of the QRS-T pattern in esophageal leads at the ventricular level with that in multiple precordial leads From the close resemblance of the QRS-T complex in Lead E_{55} to that in Lead V_6 it is evident that Lead E_{55} reflected the potential variations of the posterior diaphragmatic surface of the left ventricle The intrinsicoid deflection recorded in the P wave in Leads E_{40} and E_{35} signifies that the electrode was behind the left atrium and thereby received potentials from the left ventricular cavity transmitted through the mitral orifice and potentials from the epicardial surface of the posterobasal wall of the left ventricle near the auriculoventricular groove The fact that the QRS-T pattern in Lead aV_F is intermediate between that in E_{55} and E_{40} suggested that the left leg lead reflected a mixture of potentials from the posterior apical and posterior basal aspects of the left ventricle These considerations governed the placement of the electrode in reference to the sagittal section of the left ventricle and atrium in Fig 7

The registration of the QRS complex is analyzed in Fig 7 through a series of four diagrams prepared in the same manner as those in Fig 6 The portion of the QRS completed by the end of 02 04 06 and 08 sec is accurately reconstructed and the coincidental status of ventricular activation indicated by appropriate arrows and + and - signs

A study of the portion of the tracing recorded during the first 02 sec (Fig 7 a) reveals an initial R wave in Leads $V_{5\ 6}$ signifying early arrival of the impulse in the anterolateral aspect of the left apex and a simultaneous q wave in Leads E_{55} and aV_F, indicating that the impulse had not as yet reached and begun to activate the posteroinferior (diaphragmatic) wall of the left ventricle The downstroke in Leads E_{55} and aV_F represents the transmission of negative potentials from the [illegible] surface of the septum and the anterior wall of the left ventricle through the cavity and

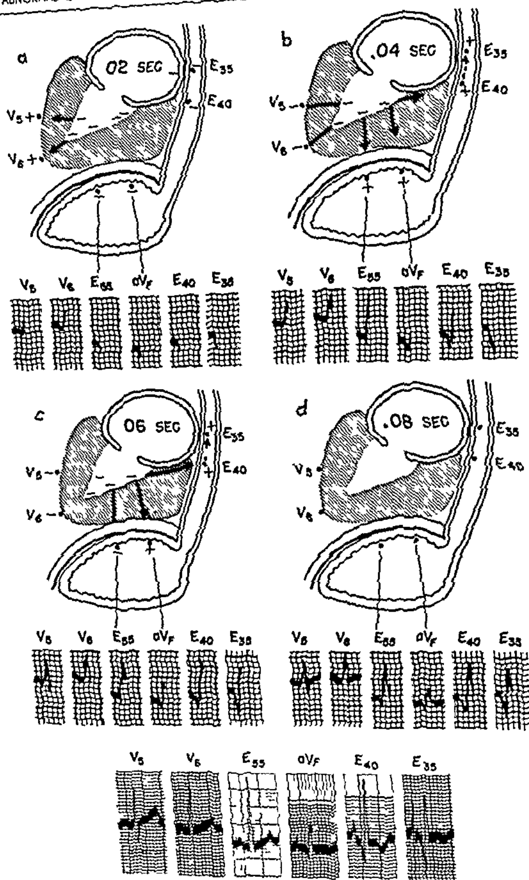

a
.02 SEC
E_{35}
E_{40}
V_5
V_6
V_5 V_6 E_{55} aVF E_{40} E_{35}
b
.04 SEC
E_{35}
E_{40}
V_5
V_6
V_5 V_6 E_{55} aVF E_{40} E_{35}
c
.06 SEC
E_{35}
E_{40}
V_5
V_6
V_5 V_6 E_{55} aVF E_{40} E_{35}
d
.08 SEC
E_{35}
E_{40}
V_5
V_6
V_5 V_6 E_{55} aVF E_{40} E_{35}
V_5 V_6 E_{55} aVF E_{40} E_{35}

Figure 7

the resting posterior wall to the stomach and left leg Immediately after the expiration of the first 02 sec the q wave at E_{55} and aV_F is replaced by an upstroke, denoting the onset of activation of the posteroinferior wall of the left ventricle The completion of the downstrokes of these q waves within 02 sec and their relatively small amplitude in comparison with the succeeding R waves indicated that the q waves of Leads E_{55} and aV_F are normal The attainment of the peak of the R wave in Lead E_{55} by the end of 04 sec and the onset of the intrinsicoid deflection immediately thereafter mark the arrival of the impulse at the epicardial surface of the posteroinferior wall of the left apex an interval well within the limits of normal Meanwhile the passage of the impulse through the anterolateral wall of the left ventricle has been completed as shown by the fact that the intrinsicoid downstroke is in progress in Leads $V_{5\ 6}$ This downward movement in Lead V_5 continues below the isoelectric line as an S wave due to transmission of negative potentials from the endocardial aspect of the posterior wall through the cavity and completely depolarized anterolateral wall to the precordial electrode

Since the registration of intrinsicoid deflections in the P waves of Leads E_{35} and E_{40} denoted levels behind the left atrium the electrode was in position to record ventricular potentials transmitted from the endocardium through the mitral orifice and left atrium as well as from the epicardium of the posterobasal wall The Q waves in Leads E_{40} and E_{35} lasted 03 sec and nearly 04 sec respectively and measured over 25 per cent and over 100 per cent of the amplitude of the succeeding R waves in the two leads When the electrode is behind the left auricle (e g in Leads E_{40} and E_{35}) Q waves are expected normally to equal or exceed those of E_{40} and E_{35} and are an expression of transmission of negative potentials from the left ventricular cavity through the mitral orifice and left auricle Thus the prominent Q waves in these leads are of no diagnostic importance The only significant temporal measurement in these leads is the time from the onset of the Q to the peak of the R wave which corresponds approximately to the time elapsing between the arrival of the impulse in the septum and the completion of its passage through the posterobasal wall of the left ventricle

Prolonged conduction through the outer wall of the left ventricle In Fig 8 the precordial electrocardiogram of a young woman with typical clinical and roentgenological signs of left ventricular hypertrophy due to hypertension is reproduced and analyzed in a manner similar to that employed in Fig 6 The QRS interval measures 11 sec and thus exceeds the customary range in normals but falls short of that in bundle branch block The transitional zone in Fig 8 like that in Fig 6 is between positions V_3 and V_4 The first three leads reflect principally the potential variations of the right side of the septum and epicardial surface of the right ventricle the last three those of the left side of the septum and epicardial surface of the left ventricle

The first drawing (Fig 8 a) represents a reconstruction of that portion of the QRS complex registered in each lead during the first 02 sec accompanied by a cross section of the ventricle showing the presumptive state of ventricular activation at the end of the period A study of this drawing reveals that the initial deflection in Leads V_1 V_2 and V_3 is upright and reaches its peak within the first 02 sec signifying early onset and completion of right ventricular activation similar in every respect to that found in normal tracings reproduced in Fig 6 The initial upstroke in Lead V_4 facing the anteroseptal wall of the left ventricle signifies early arrival of the impulse in that region in this case as in the normal An initial R wave is also recorded in Lead V_5 The fact that the portion of the R wave recorded in the first 02 sec is slightly smaller at position V_5 than at V_4 is presumably referable to a slightly later arrival of the impulse in the subendocardial muscle beneath position V_5 The delay in arrival of the impulse in the portion of the lateral wall subtended by the electrode at position V_6 is sufficient to permit the registration of an initial downstroke reflecting cavity potentials transmitted through the as yet unactivated lateral wall Since the downstroke of the q wave in Lead V_6 is only 02 sec in duration and is much less than 25 per cent of the amplitude of the succeeding R wave this q wave is entirely normal and quite comparable to that in the corresponding lead of Fig 6

The striking feature of the next two drawings representing the state of activation at the end of 04 and 06 sec respectively is the increased duration and amplitude of the R wave in left

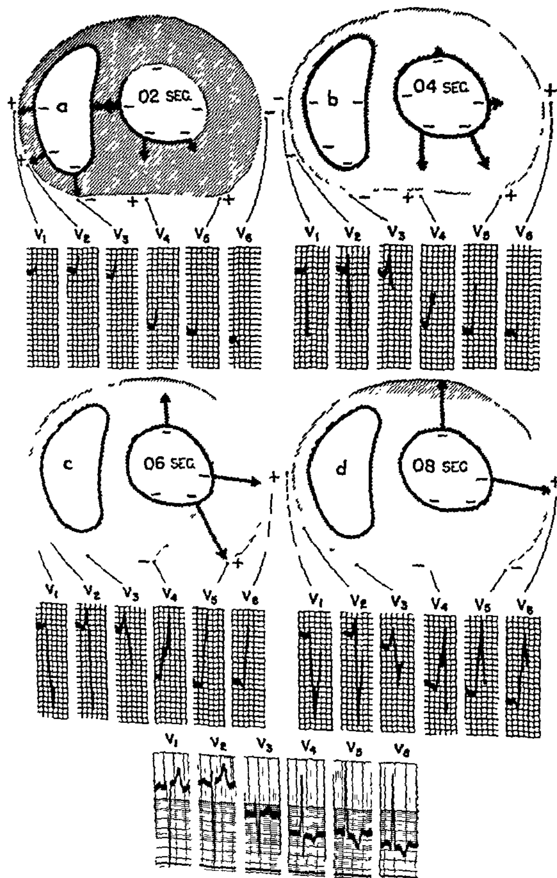


Figure 8

ventricular leads in comparison with the normal findings as reproduced in drawings at similar time intervals in Fig 6 The string is still moving upward at the end of 04 sec in the records taken at positions V_4 V_5 and V_6 indicating that the impulse is still in progress through the left ventricular wall as shown by the arrowhead in the second drawing The attainment of the peak of the R wave marking the arrival of the impulse at the epicardial surface requires a total of 05 06 and 07 sec from the beginning of the QRS complex in Leads V_4 V_5 and V_6 respectively of which 05 sec is consumed in recording the upstroke of the R wave The increased time interval from the onset of the Q wave to the peak of the R (and from onset to peak of R) coupled with the increased amplitude of the R wave in leads over the left ventricle differentiate the QRS complex of this case from the normal illustrated in Fig 6 and are indicative of prolonged activation of the outer wall of the left ventricle In patterns of this type left ventricular hypertrophy is almost always present but a myocardial lesion prolonging conduction in the absence of hypertrophy is occasionally responsible The increased thickness of myocardium which must be traversed in left ventricular hypertrophy accounts for the longer time consumed and the greater voltage developed

The lengthened interval required for left ventricular activation makes a longer time available for the transmission of the negative potentials of the left ventricular cavity through the septum and the right ventricular wall Thus the intrinsicoid movement in right ventricular Leads V_1 V_2 and V_3 continues downward as an exceptionally deep S wave which like the tall R wave in left ventricular leads is a feature of left ventricular hypertrophy A small but definite S wave is registered in V_4 due to continuing activation of other portions of the left ventricle after completion of depolarization of the anteroseptal wall The absence of an S wave from Leads V_5 and V_6 is in keeping with late completion of activation of the lateral wall of the left ventricle

The slurring or notching of the ascending limb of the R wave in Lead V_4 and of the descending limb of the S wave in V_3 are common findings in leads near the transitional zone and may reflect the arrival of the impulse at the epicardial surface of the anterior terminus of the septum or the opposite ventricle The slurring of the downstroke of the S wave in V_3 is synchronous with the peak of the R wave in V_4 and apparently marks the arrival of the impulse at the epicardial surface of the anteroseptal wall of the left ventricle The slurring of the upstroke of the R wave in V_4 occurs later than the peak of the R wave in right ventricular Leads V_1 V_2 and V_3 but earlier than the peak of the R wave in left ventricular leads and may be due to arrival of the impulse at the epicardium covering the anterior terminus of the intervening septum

The depression of the S-T junctions and deep inversion of the T waves in left ventricular Leads V_4 V_5 and V_6 and the reciprocal S-T and T patterns in right ventricular Leads V_1 and V_2 are characteristic manifestations of this pattern The prolonged activation of the outer wall of the left ventricle is apparently accompanied by prolonged repolarization The direction of the S-T and T in left ventricular leads indicates negativity of the subjacent surface and subepicardial layer of the left ventricle throughout repolarization and is consistent with progress from endocardium to epicardium – the reverse of the normal process The depression of the S-T junction and segment suggests that repolarization is in progress at the completion of the QRS and the convexity of the S-T together with the inversion of the T wave and lengthening of the Q-T interval are in keeping with late and prolonged repolarization of the subepicardial zone

Left bundle branch block In Fig 9 the electrocardiogram of a patient with advanced hypertensive heart disease is reproduced and analyzed by means of a set of four diagrams which reconstruct the QRS complex as registered during the first 02 04 08 and 12 sec respectively The diagram of the ventricles differs from that of Figs 6 and 8 in that a coronal rather than a transverse section is employed in order to depict the bundle of His and its right and left branches The transitional zone is situated between the V_4 and V_5 positions so that the first four precordial leads reflect principally the potential variations of the epicardial surface of the right ventricle the last two leads those of the left ventricle

Comparison of Leads V_1 V_2 V_5 and V_6 with the corresponding leads of a patient with typical electrocardiographic findings of left ventricular hypertrophy (Fig 8) reveals a superficial

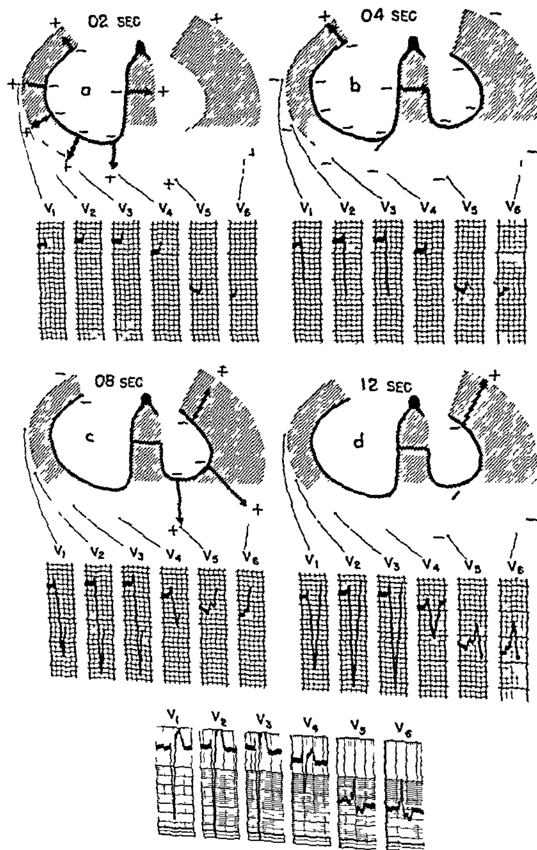


Figure 9

resemblance with respect to the relative amplitudes of the R and S waves the displacement of the S-T junctions and the direction of the T waves The most *significant differences* between the two tracings will be found in the duration of the QRS complex and in the contour of this complex in Leads V_5 and V_6 These features require detailed consideration

The QRS interval of 13 sec in association with a sinus rhythm and P-R interval exceeding 12 sec establishes the presence of an intraventricular conduction defect A comparison of the time interval preceding the onset of the intrinsicoid deflection in leads over the right with that in leads over the left ventricle reveals the ventricle through which conduction is delayed The fact that a mere 02 sec is required for the attainment of the peak of the R wave in the first four precordial leads and for the inscription of the intrinsicoid deflection in Lead V_1 signifies early onset and completion of right ventricular activation Hence the lesion responsible for the prolongation of the QRS interval is not located in the right ventricle On the other hand the precipitous downstroke representative of the intrinsicoid deflection in Leads V_5 and V_6 does not begin until 08 sec after the onset of the QRS complex indicating an abnormal delay in arrival of the impulse at the epicardial surface of the anterolateral wall of the left ventricle Therefore the prolongation of the QRS interval is caused by a conduction defect in the left ventricle

The decision as to whether the conduction defect is in the septum or free wall of the left ventricle is made from the direction of the initial phase of the QRS complex together with the contour of the rest of the tracing preceding the delayed intrinsicoid deflection If the prolongation of the QRS interval in this tracing were the result of a conduction defect limited to the outer wall of the left ventricle, Leads V_5 and V_6 should have displayed a Q wave representing negative cavity potentials derived from impulses passing from left to right through the septum and transmitted to the left axilla up until the beginning of activation of the anterolateral wall Furthermore if the conduction delay were referable to a lesion in the subendocardial layer of the anterolateral wall an abnormally deep and prolonged Q wave should have been present as an expression of the longer interval between the spread of the impulse through the septum and its arrival in responsive muscle in the free wall This situation will be exemplified in Fig 19 If the lesion of the outer wall spared the subendocardial layer but involved the midzone a normal Q wave followed by a notched or slurred upstroke would have been expected A Q wave preceding a notched or slurred R wave in leads facing the epicardial surface of the left ventricle would be interpreted as evidence (1) that the septum was activated in the normal fashion, (2) that the conduction defect responsible for the distortion of the R wave was located in the outer wall

The initial and late upstrokes with intervening notching or slurring in V_5 and V_6 of Fig 9 are distinct from the foregoing patterns The registration of an initial R rather than a Q wave in all precordial and unipolar limb leads with a late intrinsicoid deflection (left ventricular leads) indicates early positivity of the left ventricular cavity due to septal activation by impulses passing from right to left as shown by the arrow in Fig 9 a The secondary upstroke or R' deflection beginning at the nadir of the notch 04 sec after the onset of the QRS complex marks the arrival of the impulse in the outer wall of the left ventricle Septal activation from right to left together with abnormal delay in arrival of the impulse in the outer wall of the left ventricle establishes the presence of left bundle branch block The time elapsing between the onset of the QRS and the onset of the R' deflection serves as an index of the time consumed in septal activation An interval of 04 sec or more signifies complete left bundle branch block an interval between 02 and 04 sec is associated with incomplete left bundle branch block

The time elapsing between the onset and peak of the secondary upstroke or R' deflection in V_5 and V_6 constitutes a rough index of the time consumed in passage of the impulse from the endocardial to the epicardial surface of the anterolateral wall of the left ventricle The 04 sec interval in this case is borderline and not indicative of prolonged activation of the underlying left ventricular wall The registration of distinct S waves following the intrinsicoid deflections in Leads V_5 and V_6 constitutes evidence of continuing activation of more lateral or posterior portions of the left ventricle after completion of depolarization of the anterolateral wall as depicted in Fig 7 It is therefore probable that leads from the esophagus or back would have shown an abnormally

prolonged R' upstroke representative of hypertrophy and/or delayed conduction in the posterior or lateral wall Activation of the free wall of the left ventricle not only produces the secondary upstroke in Leads V_5 and V_6 but also contributes to the deep broad S waves simultaneously registered in right ventricular leads (Fig 9 c)

Thus a diagnosis of left bundle branch block is reached in Fig 9 through the demonstration of (1) a normal P wave and P-R interval of 12 sec or more signifying that the impulse which has caused the QRS complex was of auricular origin and reached the ventricles through the auriculo-ventricular node (2) a QRS interval of 12 sec or more which denotes a conduction defect somewhere in the ventricles (3) an early attainment of the peak of the R wave in right ventricular leads and an abnormally late attainment in left ventricular leads indicating that the spread of the impulse was normal through the right ventricle and delayed through the left ventricle and (4) an initial upright deflection in left ventricular leads signifying that the conduction defect was in the left bundle branch rather than in the outer wall of the left ventricle The depression of the S-T junctions and inversion of the T waves in left ventricular leads and the elevated S-T junctions and upright T waves in right ventricular leads constitute the usual findings in left bundle branch block and presumably have an analogous genesis to those in Fig 8 The S-T displacement in this case was exaggerated as a result of digitalis action

In Fig 10 the mechanism of registration of the QRS-T in the normal electrocardiogram and in left bundle branch block is contrasted by means of a reconstruction of the QRS-T at fixed intervals after its onset along with diagrams of the probable electrophysical state of the ventricles In the diagrams polarized muscle is stippled activated muscle is left blank and the vector associated with the process of activation is represented by an arrow

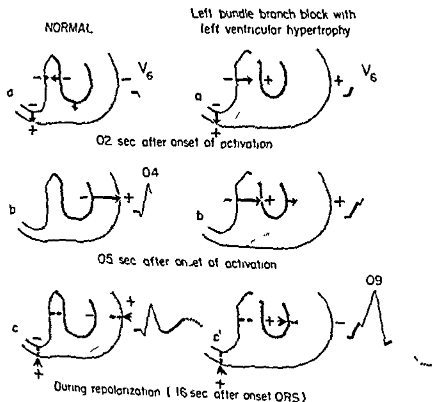


Figure 10

During the first 02 sec after the onset of the QRS a q wave is recorded in Lead V_6 of the normal representing negative potentials derived from left to right activation of the septum and

transmitted to the axilla prior to the arrival of the activating impulse in the lateral wall (Fig 10 a) *Meanwhile* an R wave is recorded in Lead V_6 in left bundle branch block due to activation of the septum in a right-to-left direction as illustrated in Fig 10 a'

The middle pair of diagrams depict the *portion of the QRS registered in* V_6 during the first 05 *sec* along with the progress of activation *In the normal the* q wave has been *succeeded by* an upstroke derived *from activation of the free wall of the left* ventricle and recognized as normal by its contour and duration Prior to the *expiration of 05 sec* in the normal the peak of the R wave has been passed and the *succeeding intrinsicoid downstroke is in* progress reflecting extinction of potentials arising *from activation of* the underlying wall Meanwhile an initial R wave and a notch, followed by *the beginning of an R' wave* have *been recorded in Fig 10* b, reflecting *right-to-left* activation of the septum during the *first 04 sec to produce* the *R* wave then onset of activation of the outer wall of the left *ventricle to initiate the R' wave* The *total* time elapsing *from onset to* completion of the R' wave *covers the interval* between 04 to 09 sec, as illustrated in Fig 10 b' and 10 c' and reflects *prolonged* conduction through the free wall of the left ventricle probably from the depicted *hypertrophy*

The lower pair of diagrams exemplify *the progress of repolarization* The registration of an upright T wave in the normal (*Fig 10 c*) *indicates positivity* of the surface and is in keeping with early repolarization *of the subepicardial layer and progression* centripetally The registration of a *convexly inverted* T *wave in left bundle branch block (Fig 10 c') indicates negativity* of the surface and is in keeping *with centrifugal repolarization* and late recovery of the *subepicardial layer*

Fig 11 exemplifies the registration of the QRS complex in incomplete and complete left bundle branch block *In each case* the initial upstroke *in* V_6 which would also be recorded from the left

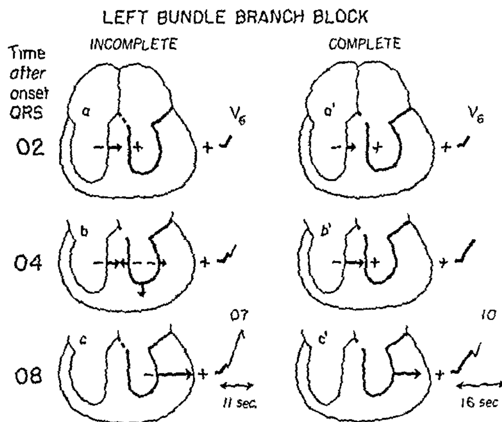


Figure 11

ventricular cavity indicates a right-to-left septal vector as depicted in the upper pair of diagrams Septal activation requires 04 sec or more in complete left bundle branch block (Fig 11 b') and between 02 and 04 sec in incomplete left bundle branch block (Fig 11, b) The beginning

of the R' deflection in V_6 marks the arrival of the impulse in the subjacent wall and the peak marks its extinction The intervening period serves as an index of the time required for activation of the free wall of the left ventricle and is prolonged in hypertrophy and/or aberrant activation of the subjacent outer wall The total time from onset of the QRS to the R' peak is prolonged to 08 sec or more in complete left bundle branch block and to between 06 and 08 sec in incomplete left bundle branch block (Fig 11 c and c')

Fig 12 illustrates a relatively common finding characterized by the registration of (1) an rR' complex in Lead V_6 and in all other leads at the horizontal level of the apex of the left ventricle ($V_{7\ 8}$ and usually V_5 and V_4) (2) a qR complex in Lead HV_6 and in all leads at the same horizontal level overlying the base of the left ventricle (HV_7 HV_8 and usually HV_5) The upright initial deflection in V_6 and in all leads facing the apex of the left ventricle points to a right-to-left vector in activation of the apical half of the septum the downward initial deflection in HV_6 and in all leads facing the base of the left ventricle is an expression of the usual *left-to-right vector in activation* of the basal half of the septum This combination of findings would be expected from the fact that (1) in normal dogs onset of activation in the left side of the septum precedes that in the right by a very brief interval at the apex and by a significantly longer interval at the base (2) lesions of the left side of the apical portion of the septum are commoner than lesions elsewhere in the septum

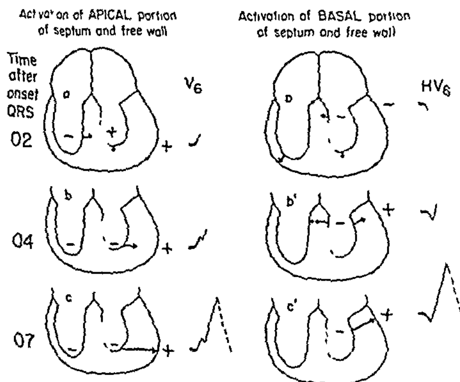


Figure 12

An interval of 02 to 04 sec between the onset of the initial R and the succeeding R' in Lead V_6 and in all other left ventricular leads at the horizontal level of the apex along with a total QRS duration of 10 to 11 sec is in keeping with incomplete left bundle branch block The concomitant registration of a qR complex in HV_6 and other leads facing the base of the left ventricle suggests that the incomplete left bundle branch block and its causative lesion are confined to the apical portion of the septum (Fig 12) A normal q wave in HV_6 and corresponding leads (less than 03 sec in duration and less than 25 per cent of the amplitude of the succeeding R wave) excludes an infarction of the subendocardial portion of the base of the outer wall of sufficient magnitude to

significantly delay the onset of activation of this portion of the heart * An upstroke derived from the free wall (R of a qR complex or R' wave of an rR' pattern) that exceeds 04 sec from onset to peak is an index of prolonged conduction usually due to hypertrophy

In Fig 13, the patterns of left bundle branch block and conduction defect in the outer wall of the left ventricle are contrasted *Both are produced by an impulse arising in the auricle* and transmitted to the ventricles through the auriculoventricular node Both are characterized by broad slurred, or notched QRS complexes 12 sec or longer in duration Differentiation is made on the basis of the direction of the initial deflection in left ventricular leads In left bundle branch block there is an initial R wave in all left ventricular leads as the result of a right-to-left septal vector (Fig 13, a) With an intact septum and a conduction defect in the free wall of the left ventricle, there is an initial Q (Fig 13, a'), the amplitude and duration of which will depend upon the amount of delay in onset of activation of the free wall of the left ventricle The upstroke of the R wave is *frequently slurred or notched* presumably reflecting aberrance in the propagation of the impulse through the pathologic outer wall of the left ventricle (Fig 13 c')

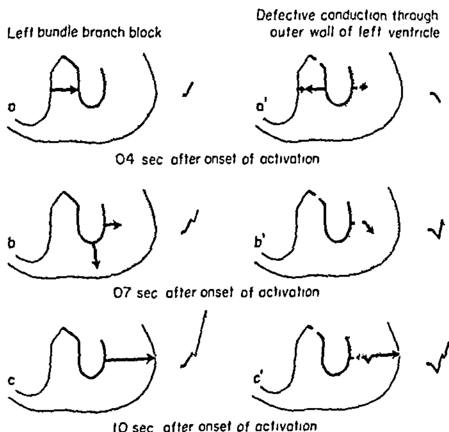


Figure 13

Prolonged conduction through the outer wall of the right ventricle In Fig 14 the precordial electrocardiogram of a young woman with typical clinical and roentgenologic signs of right ventricular hypertrophy due to mitral stenosis is reproduced and analyzed in a manner similar to that employed in Figs 6 and 8 The duration of the QRS complex is 10 sec A quick perusal of the tracings shows a striking difference from the normal and an approximate reversal of the pattern of left ventricular hypertrophy depicted in Fig 8 The tracings recorded at positions V_1 and

*An abnormal QR complex would be expected in HV_6 in the presence of infarction of the subendocardial portion of the base of the lateral wall when the proximal part of the septum is activated in a left to right direction

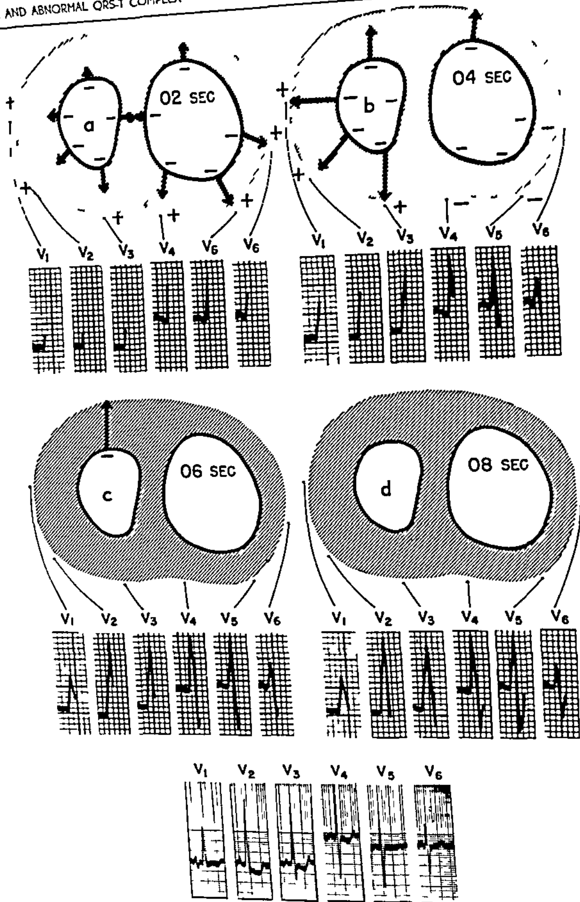


Figure 14

V_2 in this case resemble those obtained at positions V_6 and V_5 in left ventricular hypertrophy whereas the pattern in Leads V_6 and V_5 of this case is more representative of that customarily found in Leads V_1 and V_2 The general resemblance of the QRS complex in Lead V_3 of Fig 14 to that of V_2 and V_1 suggests that the precordial electrode at the V_3 position was dominated by right ventricular potentials, and the general resemblance of the QRS of V_4 to that of V_5 and V_6 suggests that the precordial electrode in this location was dominated by left ventricular potentials Further details are brought out through a study of the portions of the QRS completed in each lead at the end of 02, 04 and 06 sec, respectively

The initial deflection is upright in Leads V_6 and V_5 as well as in V_4 of this case, indicating early activation of the lateral as well as the anterior aspect of the left ventricle Furthermore, the peak of the R wave is attained in leads over the left ventricle in the very short span of 02 sec presumably due in part to early arrival of the impulse at the epicardial surface of the anterior and lateral walls of the left ventricle, in part to opposing forces transmitted from the continuing activation of the right ventricle

The initial deflection in Leads V_2 and V_3 is upright but the QRS complex in V_1 apparently begins with a minute downstroke which is quickly replaced by an R wave Two explanations for these findings were considered (1) the right ventricular cavity was initially electropositive from activation of the septum in the usual left-to-right direction The potentials transmitted from the right side of the septum would be expected to cause initial upstrokes in leads over the right ventricular epicardium near the septum (V_3 and V_2) but may dwindle sufficiently to permit the registration of a downstroke in Lead V_1 and leads farther to the right presumably derived from left ventricular cavity potentials transmitted through the left and right atria (2) the right ventricular cavity was initially electronegative from activation of the septum from right to left an unusual vector that has been conclusively demonstrated in some cases of right ventricular hypertrophy by recordings during right ventricular catheterization If such a vector were present in this case, the registration of an initial upstroke in Leads V_3 and V_2 facing the anteroseptal wall of the right ventricle would indicate early onset of activation of the underlying subendocardial layer whereas the q wave in Lead V_1 facing the anterolateral wall of the right ventricle would indicate sufficient delay in arrival of the impulse to permit momentary transmission of cavity potentials to the surface Although no final conclusion was reached in this case because of the lack of cardiac catheterization the influence of the direction of the septal vector on the configuration of the QRS in right ventricular hypertrophy will be presented in Fig 15

The most striking features of Leads V_1 V_2 and V_3 are the unusually tall R wave the late onset of the intrinsicoid deflection and the small or absent S wave From a study of the portion of the tracing completed in the first 02 sec (Fig 14 a) it would appear that the impulse has reached the epicardial surface of the lateral wall of the left ventricle as shown by the attainment of the peak of the R wave in Leads V_5 and V_6 but only activated a fraction of the right ventricular wall as shown by the relatively small portion of the R wave registered in Leads V_1, V_2 and V_3 The continuing upstroke in right ventricular leads during the next 02 sec (Fig 14 b) and the requirement of a total of 04 to 05 sec for attainment of the peak of the R wave in these leads are typical of prolonged conduction through the outer wall of the right ventricle The basis is usually right ventricular hypertrophy occasionally a myocardial lesion prolonging conduction in the absence of demonstrable hypertrophy

The registration of the descending limb of the S wave in Leads V_5 and V_6 synchronously with the upper portion of the ascending limb of the R wave in V_1 and V_2 points to a common origin and suggests that the S wave is due to transmission of negative right ventricular cavity potentials through the depolarized septum and anterolateral walls of the left ventricle to the left axilla The presence of a small S wave in Lead V_3 facing the anteroseptal wall of the right ventricle together with its absence from V_1 would indicate completion of activation of the anteroseptal wall of the right ventricle sufficiently ahead of the lateral wall to permit brief transmission of negative cavity potentials to the surface

Although the contour of the S T segment in Leads V_2 and V_3 suggests digitalis action the patient had received no cardiac glycosides The depressed S-T junctions and inverted to diphasic T waves in Leads V_1 V_2 and V_3 are an expression of delayed repolarization of the subepicardial layer of the right ventricle secondary to prolonged activation and thus are analogous to those over the hypertrophied left ventricle reproduced in Fig 8

Upon pathologic examination of right ventricular hypertrophy three zones can usually be distinguished on the basis of myocardial thickness (1) the tricuspid ring and/or crista supraventricularis in which the myocardium equals or exceeds the thickness of the normal left ventricle, (2) the trabeculated body of the right ventricle which is usually dilated but relatively thin (3) the normal left ventricle In the coronal diagram of Fig 15 the electrode at V_{3R} reflects chiefly the potential variations of the hypertrophied tricuspid ring that at V_3 reflects mainly those transmitted from the relatively thin dilated body of the right ventricle whereas that at V_6 faces the normal left ventricle

The direction of the initial deflection of the QRS depends upon the septal vector The more frequent findings in these three representative leads together with diagrams depicting the probable state of ventricular activation are reconstructed in the left half of Fig 15 The initial upstroke recorded in right ventricular Leads V_{3R} and V_3 and the synchronous downstroke in V_6 indicate a left to right septal vector as shown in the diagram of activation during the first 01 sec The continuing ascent of the R wave in V_3 between 01 and 02 sec and the simultaneous downstroke in V_{3R} are derived from activation of the body of the right ventricle before arrival of the impulse in the more distant tricuspid ring Two channel records reveal simultaneous positivity of the epicardial surface of the body of the right ventricle and negativity of the cavity as shown in the diagram labelled 02 sec Meanwhile the brief q wave in V_6 has been replaced by an R wave signifying activation of the anterolateral wall of the left ventricle A prominent upstroke in V_{3R} and a reciprocal S wave in Leads V_3 and V_6 are recorded late (between the intervals of 02 sec and 06 sec in the diagram) as a result of late activation of the hypertrophied tricuspid ring

The QRS in Lead V_{3R} thus comprises r and R' deflections separated by notching or slurring and thus resembles in shape the rR complex found in the corresponding leads in incomplete right bundle branch block The distinction is based upon the duration of the initial r wave of septal origin The time elapsing from the onset of the QRS to the onset of the R' deflection should exceed 02 sec in incomplete right bundle branch block and should usually measure between 03 and 04 sec with normal septal activation it should not exceed 02 sec This *measurement* is made in the lead to the right of the septum with the best developed initial component of an rR' or rRs complex

The serial tracings on the right half of Fig 15 represent relatively uncommon but important alternative findings in right ventricular hypertrophy The initial downstroke in Leads V_{3R} and V_3 and the synchronous R wave in V_6 indicate a right to left septal vector In records from the right ventricular cavity in such cases the presence of an initial downstroke rather than the usual r wave has been demonstrated thereby confirming the leftward direction of the septal vector illustrated in the 01 sec diagram The continuing descent at V_{3R} together with the interruption in V_3 by an upstroke that may cross the isoelectric line or may not reach it (as illustrated in the 02 diagram) are referable to activation of the body of the right ventricle before arrival of the impulse in the more distant tricuspid ring The registration of a prominent late R wave in V_{3R} and a reciprocal S wave in V_3 and V_6 is an expression of late activation of the hypertrophied tricuspid ring as illustrated in the 06 sec diagram

The characteristics of the qR pattern transmitted from the tricuspid ring the notched QS from the body of the right ventricle and the RS from the left ventricle are important in the differentiation from the qR complexes registered from the right precordium and the QS complexes recorded over the anterior wall of the left ventricle in the presence of anteroseptal infarction with right bundle branch block to be discussed later

Right bundle branch block The precordial electrocardiogram of a patient with long-standing femoral arteriovenous aneurysm is reproduced in Fig 16 An attempted closure nine years previously was unsuccessful and further surgery was refused Serial limb leads over this period showed a first degree auriculoventricular block and no significant changes in the QRS-T pattern At autopsy, marked right ventricular hypertrophy and moderate left ventricular hypertrophy were demonstrated

RIGHT VENTRICULAR HYPERTROPHY

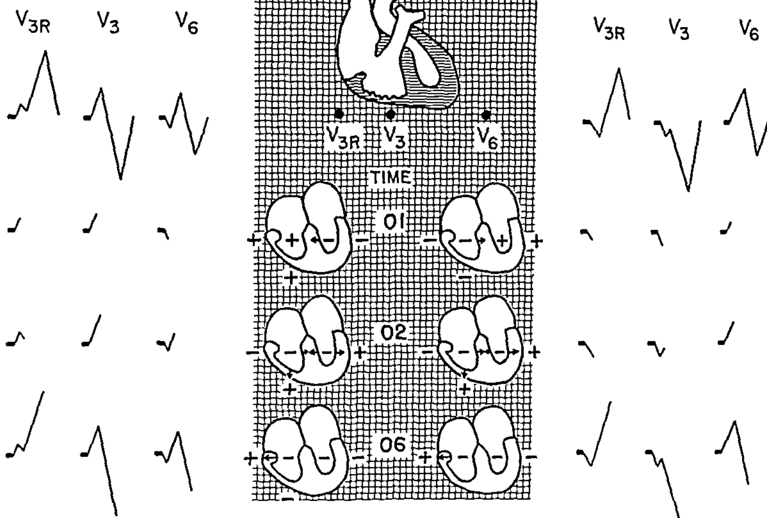


Figure 15

The tracing is analyzed in a series of four diagrams which reconstruct the QRS complex as it was registered in each lead during the first 02 sec 04 sec 08 sec 12 sec respectively In these diagrams as in Fig 9 a coronal section of the ventricles is employed in order to depict the bundle of His and its right and left branches

A sinus rhythm and a constant P-R interval of 28 sec without dropped beats were present The QRS interval of 16 sec was the most striking feature of the tracing and in view of the rhythm established the presence of an intraventricular conduction defect

A study of the R/S relationships in the six precordial leads reveals that the broad double peaked R and narrow S waves recorded in V_1 and V_2 contrast sharply with the narrow R and wide slurred S waves in V_3 V_4 V_5 and V_6 Thus the transitional zone is situated between the V_2 and V_3 positions the first two leads reflecting principally the potential variations of the epicardial

surface of the right ventricle the last four leads depicting chiefly the potential variations of the epicardial surface of the left ventricle

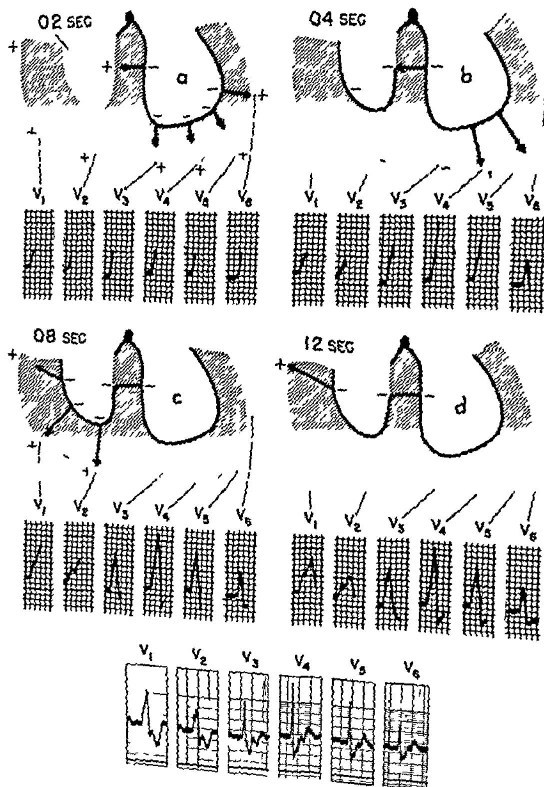


Figure 16

The initial upstroke in Leads V_3 V_4 V_5 and V_6 (Fig 16 a) together with the attainment of the peak of the R wave in these leads within 04 sec (Fig 16 b) denotes early onset and relatively early completion of activation of the anterolateral wall of the left ventricle and shows that the conduction defect responsible for the QRS prolongation is located elsewhere The fact that the intrinsicoid downstroke in Leads V_1 and V_2 begins 08 sec or more after the onset of the QRS complex places the conduction defect in the right ventricle

The nature of the defect is revealed through a study of the portion of the tracing preceding the late intrinsicoid deflection in Leads V_1 and V_2 The registration of an initial R wave in these leads indicates septal activation by impulses spreading from left to right as depicted by the arrow in Fig 16 a the secondary upstroke following a notch or coarse slurring is referable to passage of the impulse through the free wall of the right ventricle as shown by the arrow in Fig 16 c The interval of 04 sec between the onset of activation of the septum (or beginning of the initial R wave) and the onset of activation of the free wall of the right ventricle (beginning of the second R wave) is abnormally long and establishes the presence of right bundle branch block An interval of 04 sec or more may elapse between the onset and peak of the R' deflection of right bundle branch block in the absence of a grossly demonstrable lesion of the outer wall of the right ventricle at autopsy and is presumably due to aberrant activation analogous to that associated with left ventricular premature beats In this case the known right ventricular hypertrophy may have been a contributory factor An even longer time was apparently consumed in the activation of more lateral or posterior portions of the right ventricle as may be inferred from the presence of S waves in Leads V_1 and V_2

During the period between 04 and 08 sec when the R wave is being registered in V_1 and V_2 the intrinsicoid deflection is completed in Leads V_3 V_4 V_5 and V_6 and is followed by a broad slurred S wave due to transmission of negative right ventricular cavity potentials through the completely activated septum to the left precordium and axilla The opposing potentials from the hypertrophied right ventricle may have accounted for the fact that the R wave in left ventricular leads was not as great in amplitude or duration as might have been expected from the degree of left ventricular hypertrophy found at autopsy

The depressed S-T junctions and inverted T waves in right ventricular Leads V_1 and V_2 and the upright T waves in left ventricular Leads V_5 and V_6 constitute the usual findings in right bundle branch block The diphasic T waves in the intervening leads are transitional in type

In Fig 17 the mechanism of QRS registration in Lead V_1 in right bundle branch block due to septal infarction is contrasted with the uncomplicated pattern The presence of right bundle branch block in QRS complexes of supraventricular origin is shown by a duration of 12 sec or more and a late intrinsicoid deflection (delayed 08 sec or longer) in precordial leads facing the right ventricle Uncomplicated right bundle branch block displays an initial R wave from a left-to-right septal vector (Fig 17 A) and a final R' deflection from aberrant activation of the free wall of the right ventricle (Fig 17 C) These upstrokes are separated by slurring or by a downstroke of variable amplitude representing preponderance of negative potentials transmitted from the left ventricular cavity during the period between extinction of septal activation and onset of depolarization of the free wall of the right ventricle (Fig 17 B)

Right bundle branch block due to septal infarction is characterized by a QR deflection instead of an RsR' complex The registration of an initial downstroke in V_1 and other right precordial leads is referable to the septal infarct and is an expression of preponderance of negative potentials transmitted from the free wall of the left ventricle through the dead window-like infarct over positive potentials derived from activation of septal remnants (Fig 17 A') The descent of the tracing continues until the impulse makes its way by an aberrant route from the left to right ventricle, whose activation is marked by a late upstroke (Fig 17 C') Thus a QR complex is recorded composed of a Q wave of abnormal depth and duration and a late R wave

The electrocardiogram reproduced in Fig 18 was representative of a series taken over a period of six months following the advent of painless pulmonary edema in a 52-year-old diabetic woman

The presence of left bundle branch block could be established by (1) a normal sinoauricular origin and auriculoventricular conduction of the impulse (2) QRS duration of .12 sec or more (3) a broad slurred initial R wave in Leads V_5 and V_6 The depressed convex S-T segments and deeply inverted T waves in V_5 and V_6 constituted the usual findings in left bundle branch block The deep broad QS deflections in V_1 V_2 and V_3 were representative of a pattern *sometimes* recorded over the right precordium in uncomplicated left bundle branch block presumably the result of preponderance of negative potentials from the right side of the septum over positive potentials from the right ventricular wall early in the course of ventricular activation The elevated S-T segments and monophasic upright T waves in Leads V_1 V_2 constituted the expected findings in right precordial lead

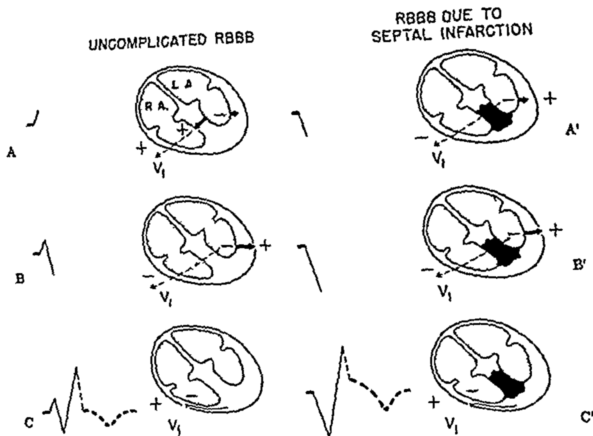


Figure 17

The QRS ... its and T waves are recorded du... T waves in $V_{5\ 6}$ indicated ele... ...ual surface of the left ventricle during diastole as illustrated in Fig 18 A and systole as in Fig 18 B The QS deflections and upright T waves in V_1 V_2 indicated electrode positions facing the epicardial surface of the right ventricle during both diastole and systole as shown in Fig 18 A B

The problem rested in the explanation for the QRS T complexes in Leads V_3 and V_4 The original interpretation of anteroseptal infarction was excluded by a meticulous post-mortem examination and it was necessary to rationalize the findings in Leads $V_{3\ 4}$ with the slight left ventricular hypertrophy found at autopsy and the left bundle branch block that existed during life The resemblance of the QS deflection in V_3 to those in V_1 and V_2 suggested that the electrode lay to the right

of the septum during diastole as indicated in Fig 18 A The Qrs pattern of relatively low voltage in V_4 could be explained as a manifestation of the transitional zone associated with an electrode position straddling the septum during diastole, depicted in Fig 18 A The Q wave corresponded to the first portion of the QS in the first three precordial leads and probably represented potentials transmitted through the anteroseptal wall of the right ventricle the R wave corresponded to the expected time of activation of the anteroseptal wall of the left ventricle and was probably derived in this manner The convex S-T segment and inverted T wave of V_4 simulated those of V_5 and V_6 and was attributable to counterclockwise rotation of the left ventricle at the beginning of mechanical systole to a position subjacent to V_4 as depicted in Fig 18 B Upon analysis of the record at V_3 the resemblance of the elevated beginning of the S-T segment to that in V_2 and the resemblance of the coved terminus of the ST-T to that in V_4 suggested mechanical rotation of the heart during registration of the complex so that the anteroseptal margin of the right ventricle was beneath the electrode at the onset of S-T and the corresponding part of the left ventricle at the end of the T, as depicted in Fig 18 B Mixed transitional zonal patterns with presumed mechanical counterclockwise rotation during systole have been encountered also in left ventricular hypertrophy without bundle branch block but with post-mortem exclusion of infarction

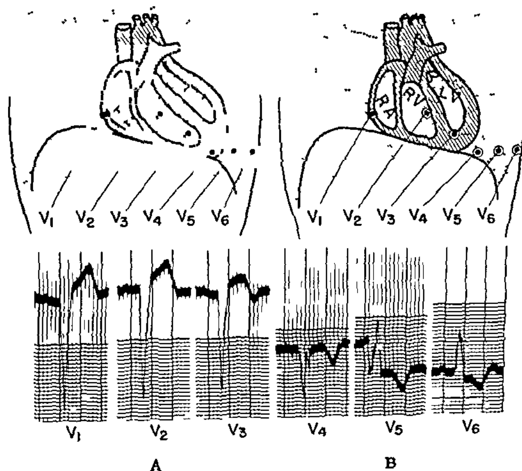


Figure 18

Direct leads in experimental myocardial infarction Since correlative studies of serial electrocardiograms with gross and microscopic pathology in animals following coronary ligation form the basis for the interpretation of electrocardiographic patterns associated with myocardial infarction in humans a brief summary of some of the more important observations in animals will be given as a prelude to an analysis of the electrocardiogram in human myocardial infarction Although the description revolves about the findings in the antero-apical portion of the left ventricle following

ligation of the descending branch of the left coronary artery comparable changes have been demonstrated elsewhere in the heart following ligation of other major branches of the coronary tree * Within a few seconds after occlusion of the anterior descending coronary artery the antero-apical portion of the left ventricle becomes pale and cyanosed and a continuous tracing from a direct lead applied to any part of the epicardium within the discolored area shows a progressive change from an upright to a sharply inverted T wave The T waves generally attain maximal depth within the first thirty seconds At this stage the S-T junctions are usually isoelectric but the S-T segments frequently exhibit the upward convexity and the T waves the sharp V-shaped inversion commonly encountered during the evolution of human myocardial infarction If the ligature is removed when the T waves have attained their maximal depth the color of the myocardium returns to normal and the T waves progressively resume their original configuration The concurrent appearance of sharp inversion of the T waves with myocardial pallor and cyanosis and the prompt disappearance of the T wave abnormalities with restoration of coronary circulation and myocardial color to normal indicate that the T wave abnormalities are the result of myocardial ischemia

If the coronary ligature is retained for more than thirty seconds progressive elevation of S-T junction is noted in direct leads from the discolored area beginning 30 to 60 sec after the onset of the occlusion and generally reaching a maximum within 60 to 90 sec At this time the S-T takeoff approaches the peak of the R wave and the T wave is monophasic upright whereas the ascending limb of the R wave is usually unaltered The elevation of the S-T junction marks the advent of the stage of acute injury Although the ischemia and subsequent injury probably extend through the entire thickness of the wall the sharp T wave inversion and subsequent S-T elevation are probably produced by the injury of the subepicardial layer as shown by the close resemblance to the ST-T pattern resulting from burns scarification and other forms of trauma strictly localized to the subepicardial portion of the myocardium On the other hand injury limited to the subendocardial layer causes depression of the S-T segment If the ligature is removed as soon as maximal S-T elevation occurs normal color returns to the myocardium the S-T junction rapidly drops to the isoelectric line the T wave meanwhile exhibiting transient inversion prior to the resumption of its original contour The complete recovery from interruption of coronary circulation for 90 sec or less suggested by return of ECG to normal is borne out by histological studies which show no evidence of myocardial lesion

It should be emphasized that the electrocardiographic abnormalities do not arise directly from the ligated coronary vessel Gradual coronary narrowing and eventual occlusion without electrocardiographic changes or histological evidence of infarction may occur spontaneously in humans and may be produced experimentally in animals when the myocardium is protected by adequate collateral circulation On the other hand myocardial ischemia injury and infarction with their characteristic electrocardiographic patterns may occur in the absence of coronary occlusion

The electrocardiographic changes which appear promptly after ligation of a coronary artery are probably an expression of chemical changes in the heart muscle secondary to deprivation of its blood supply Soon after coronary occlusion an extracellular edema begins to accumulate and within a few minutes reaches proportions recognizable by chemical and histological examination Since application of potassium compounds to the epicardium produces S-T displacement resembling that following coronary ligation it has been suggested that escape of potassium into the extracellular fluid may be responsible for the S-T elevation and the tall monophasic T waves

If the coronary ligature is retained for a longer period progressive reduction in the amplitude of the R wave and development of an abnormal Q wave may be observed in leads from the discolored area often within the first 3 or 4 minutes The abnormalities in the QRS as well as those in the S T segment and T wave are demonstrable only in leads from the area deprived of its blood supply Direct leads from other portions of the heart where normal myocardial color and

*Certain arrhythmias particularly ventricul[illegible]
complications of [illegible]
subject under [illegible] on

circulation are maintained reveal no intrinsic abnormalities in the QRS In addition to the electrocardiographic abnormalities demonstrable in the area that is deprived of its blood supply there is evidence of altered physiologic function as indicated by dilatation and decreased contractility If the coronary ligature is removed within the first 20 minutes, the myocardium may resume normal color and contractility in which event histological signs of myocardial infarction or degeneration are absent

Thus the electrocardiographic pattern customarily ascribed to myocardial infarction may be produced in animals by temporary deprivation of myocardial blood supply for periods too brief to result in myocardial infarction While a comparable situation presumably occurs in man pathologic examination of hearts from patients who survived 24 hours or more after the appearance of a typical electrocardiographic pattern has almost invariably revealed definite signs of infarction Hence the acceptance of typical QRS-T changes, to be described below as evidence of the presence of an infarct in humans is justified by the available pathologic material

Although QRS abnormalities associated with deprivation of myocardial blood supply appear before the myocardial lesion is detectable pathologically, corollative studies are best carried out after the infarction is readily recognizable The close relation of QRS abnormalities to the infarct has been demonstrated by comparison of tracings obtained by multiple direct epicardial leads with gross and histological studies of the segment of wall under each point where the exploring electrode was applied Both electrocardiographic and pathologic examination show that the area of infarction is not uniform throughout but is generally divisible into three zones namely (1) a central zone where the infarct extends through the entire wall (2) a marginal zone where the infarct involves only a portion of the thickness of the wall most commonly the subendocardial layer but sometimes the subepicardial layer or midzone (3) an ischemic zone between the boundary of the infarct and the normal muscle in which there is discoloration but no degeneration Smaller infarcts often fail to extend through the entire thickness of the wall and thus display only a marginal zone and an ischemic zone

When septal activation in the usual left-to-right vector is preserved the form of the QRS complex in a direct lead is dependent upon the relative amount of infarcted and responsive muscle in the underlying segment of ventricular wall and upon the relative locations of the infarcted and responsive muscle in reference to endocardial and epicardial surface Direct leads from points on the central zone beneath which the entire thickness of the ventricular wall is infarcted reveal a QS complex with smooth unnotched descending and ascending limbs This QS complex is similar to that simultaneously registered through an electrode inserted into the left ventricular cavity Negative potentials referred to the left ventricular cavity throughout the period of activation of the uninfarcted portions of the wall are responsible for the downstroke of the QS whereas the extinction of these electromotive forces upon completion of activation is responsible for the upstroke which returns the string to the isoelectric line A completely infarcted ventricular wall does not respond to the activating impulse but like a valvular orifice merely behaves as a window through which negative cavity potentials are transmitted to the surface to the heart If a small amount of muscle in the infarcted wall beneath the exploring electrode is responsive the descending limb of the Q wave resulting from transmission of negative cavity potentials through the infarcted area will be interrupted by a brief upward movement of relatively small magnitude due to momentary reversal of polarity of the epicardial surface from activation of subjacent muscle As soon as this small bundle of responsive muscle has been activated the downward movement of the Q wave will be resumed Thus a notch will have been produced in the descending limb of the Q wave The upstroke of the notch does not reach the isoelectric line if the electromotive force produced in the underlying wall is small, but may cross it to form an R wave if the electromotive force is larger Hence a QS complex with smooth limbs is obtained from the central zone if the underlying wall is completely infarcted whereas a QS complex with a notch in the descending limb is obtained if a small amount of myocardium is still capable of responding to the activating impulse

As the electrode is moved centrifugally in any direction a marginal zone will be reached where the infarct does not extend through the entire wall If the subendocardial layer is infarcted and the

outer portion is responsive a QR complex is obtained The initial Q wave is registered during the interval from the moment that the impulse has begun to activate the septum until it has made its way through the infarcted subendocardial muscle to reach living responsive myocardium in the subepicardial zone subtended by the electrode During this period negative potentials referred to the cavity from activation of intact portions of the septum and outer wall are transmitted through the involved wall to the overlying electrode causing a downward deflection As soon as the impulse reaches and starts to activate the responsive outer portion of the wall, the polarity of the epicardial surface is reversed and the Q wave is replaced by an upstroke The Q wave associated with subendocardial infarction is abnormal in depth and duration particularly when compared with the R wave of the same lead and as a rule greatly exceeds the normal limit of 25% of the amplitude of the R Moreover the R wave is reduced in amplitude presumably due to the failure of the infarcted subendocardial portion to contribute towards it If the infarct at the marginal zone involves the subepicardial layer or mid-portion, and spares the subendocardial layer, the initial deflection of the QRS is upright indicating early activation of the responsive subendocardial muscle however the R wave is abnormally small in amplitude reflecting the diminished magnitude of electromotive force developed in the sector beneath the exploring electrode A similar finding may be obtained with patchy infarction scattered through the wall In the ischemic zone just beyond the border of the infarct the QRS is normal in contour and therefore again corresponds to the histologic studies which show no myocardial degeneration in this area

Thus the form of the QRS in a direct lead from the epicardium serves as an index of the presence of an infarct in the underlying segment of wall and of the approximate thickness and location of the infarcted layer of muscle Through a study of the form of the QRS in multiple direct leads the approximate area of infarction may be mapped out and delineated from the surrounding normal muscle On the other hand the age of the infarct cannot be estimated accurately from a study of the QRS complex alone Abnormalities in the QRS appear before the histologic demonstrability of infarction and may persist lifelong if an appreciable amount of the myocardium is replaced by fibrous tissue however the QRS is not static throughout the course of infarction but may show changes paralleling those in the myocardium If an originally unresponsive portion of the wall recovers excitability the R wave increases in amplitude at the expense of the Q or S deflection whereas if the infarct extends further into the wall the R wave decreases or disappears

The age of the infarct is best estimated from changes in the S T segment and T wave in serial electrocardiograms Classical serial changes in the S T segment and T wave are demonstrable during the first few weeks after infarction by direct leads from any portion of its surface except from areas where the underlying muscle is entirely dead In such areas the T wave like the QRS, resembles that obtained by a direct lead from the ventricular cavity The QRS consists of a QS complex with smooth descending and ascending limbs and the T wave is generally upright and thus opposite in direction to the inverted T recorded in epicardial leads from the zone of ischemia When an R wave is present signifying activation of some portion of the underlying wall the S-T segment and T wave reflect repolarization of this muscle the dominant influence probably coming from the subepicardial surface Even though the subepicardial muscle is capable of responding to the stimulus it will usually exhibit abnormal repolarization for a considerable time after the onset of the infarction Initially the S T junction is elevated and the T wave monophasic upright reflecting injury to the subepicardial myocardium The S-T elevation is an indirect effect of a potential difference which exists between the injured and uninjured portions of the myocardium throughout the entire cycle except at the end of the QRS The injured area is electronegative in relation to the remainder of the ventricle throughout diastole and until completion of activation (i e until the end of the QRS) when the potential difference is momentarily abolished The relative negativity of the epicardial surface of the injured area throughout diastole causes a depression of the entire tracing between the T wave and the S-T junction of the next cycle Although the actual effect of injury of the subepicardial myocardium is to depress the isoelectric line in reference to the S T junction it is customary to describe the resultant tracing as an elevation of the S T junction in reference to the isoelectric line This will be exemplified and elaborated in Fig 21

During the course of the first few days after the onset of the infarction the elevated S-T junction gradually approaches the isoelectric line, the terminal portion of the T wave meanwhile dipping below the isoelectric line By the time the S-T junction reaches the isoelectric line the T wave has become deeply inverted This marks the disappearance of the pattern of acute subepicardial injury and its replacement by the pattern of subacute subepicardial injury The T wave may become still more deeply inverted over the next one to three weeks and then gradually decreases in depth over the next few weeks or months and finally may become upright and normal in contour with complete recovery of the ischemic muscle The age of the infarct is thus judged from the evolution of the S-T segment and T wave in leads which exhibit an abnormal QRS complex due to infarction Meanwhile in the original zone of ischemia just beyond the border of the infarct the initially inverted T waves tend to revert to normal much more rapidly

The principal differences between direct and precordial (or esophageal) leads have been discussed previously and will merely be reviewed briefly insofar as they have bearing on electrocardiographic changes produced by infarction The major difference is referable to the fact that the tracing obtained by precordial leads is dominated by the potential variations of a much larger surface of epicardium than that obtained by a direct lead If the infarct is uniform throughout the area which has the dominant influence on the precordial lead the form of the QRS of the precordial lead will simulate that in direct leads from the underlying epicardium Thus the registration of an unnotched QS in a precordial lead over the left ventricle would suggest the presence of a relatively large area of infarction extending through the entire wall The presence of a rather uniform QR complex in contiguous precordial leads would suggest a rather extensive subendocardial infarct If the infarct is irregular in distribution the tracing obtained by precordial lead will represent a mean of those obtained by multiple direct leads from the underlying epicardium If the central zone is small direct leads may reveal a QS complex with smooth descending and ascending limbs whereas the overlying precordial lead may register a notched QS complex or a QR due to admixture of effects from the central and marginal zones If the infarct is very small and limited to a portion of the wall multiple direct leads may yield diagnostic signs whereas precordial leads may be equivocal due to admixture of effects from infarcted and surrounding normal muscle While direct leads tend to show a sharp delineation between central marginal and ischemic zones precordial leads show a more gradual transition due to overlapping effects Thus precordial leads do not provide as accurate an index of the thickness of the wall infarcted or of the total surface area of the infarct as do direct leads Nevertheless multiple precordial leads together with esophageal leads will furnish sufficient data for the diagnosis of all but the very small infarcts and will permit a sufficiently accurate localization and estimate of size for clinical purposes

Anterolateral myocardial infarction Fig 19 presents an analysis of the precordial electrocardiogram obtained on a 35-year-old woman approximately 2 years after an acute myocardial infarction and about 6 months prior to death Autopsy revealed a completely healed anterolateral infarct occupying the area left unshaded in the diagram The electrocardiogram is analyzed through a series of four drawings which reconstruct the portion of the QRS complex registered in each lead during the first 02 04 06 and 08 sec respectively

Leads at either end of the precordium show contrasting patterns the rS deflection in Lead V_1 representing chiefly the potential variations of the epicardial surface of the right ventricle the qR complex in V_6 reflecting the potential variations of the lateral wall of the left ventricle The sharp intrinsicoid downstroke in the upright P wave recorded in Lead V_1 suggests that the electrode was in the vicinity of the tricuspid ring The minute initial R wave preceding the deep broad S wave in Lead V_2 indicates that the electrode was still on the right ventricular side of the septum The QS deflection in Lead V_3 was transitional between the rS patterns in V_1 and V_2 and the QR patterns in V_4, V_5 and V_6, suggesting that the electrode was near the septum but probably on the left ventricular side Although Leads V_4 V_5 and V_6 differ in respect to QR ratio they show a synchronous late intrinsicoid deflection of left ventricular origin Leads V_5 and V_6 of this case are comparable to the corresponding leads of Fig 9 in prolongation of QRS interval to 12 sec and in the delay in onset of the intrinsicoid deflection to 08 sec , but differ significantly in respect to the direction of

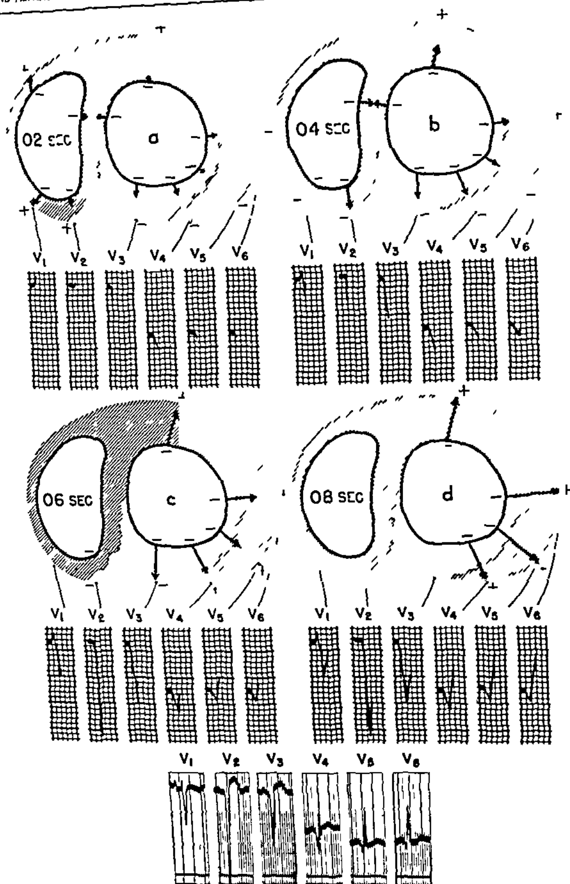


Figure 19

the initial deflection The significance of the latter in localizing the conduction defect will be brought out subsequently in the analysis of the tracing

In Lead V_1 the initial deflection is upright and rises 3 5 mm to reach its peak by the end of 02 sec This represents a normal finding for precordial leads overlying the *right ventricle and* is in keeping with the demonstration of a normal anterolateral right ventricular wall at autopsy Attention is directed to the marked reduction of the R wave in Lead V_2 in comparison with its counterpart in V_1 As the electrode is moved from the V_1 to the V_2 position an increase in the amplitude of the R wave is expected because of closer proximity of the electrode to the septum *and left ventricle The decrease is an abnormal finding* and may occur under the following circumstances (1) infarction of septum and/or anteroseptal wall of the left ventricle with consequent loss of positive potentials transmitted from this *portion of the heart to the neighboring* V_2 position, (2) disproportion between the muscle mass at the tricuspid orifice (near position V_1) and that forming the anteroseptal wall of the right ventricle (near position V_2) due either to preponderant hypertrophy of the muscular tricuspid ring or to infarction or other destructive lesion of the anteroseptal aspect of the right ventricle The former alternative is responsible for the reduction *in the initial R* wave in Lead V_2 of this case, as is shown by the abnormal Q patterns in precordial leads over the left ventricle and subsequently confirmed at autopsy

A Q wave is recorded in left ventricular leads V_3, V_4 V_5 and V_6 (Fig 19 a), indicating initial negativity of the left ventricular cavity due to early activation of the left side of the septum by *impulses distributed through the left Purkinje* plexus This finding excludes uncomplicated left bundle branch block as a cause of the delayed intrinsicoid deflection in the last three precordial leads and the *prolongation in the QRS interval Although Q waves may rarely be recorded in left* ventricular leads when left bundle branch block is complicated by almost complete infarction of the septum this possibility is excluded in the present case by the fact that (1) the QRS interval is only 12 sec (2) the Q wave is longer in duration in left ventricular leads V_3 and V_4 which are relatively near the right ventricle than in V_6 which is far removed

A study of Fig 19 b shows that the initial downstroke continues beyond the normal limit of 02 sec in each of the last four precordial leads In Lead V_3 the tracing describes a smooth downward course for at least 06 sec then returns to the isoelectric line thereby completing an unnotched QS deflection (Fig 19 c and d) The lack of an initial or final R wave together with the absence *of a notch on either limb of this QS deflection would suggest that the anteroseptal* wall of the left ventricle subtended by the electrode at V_3 is unresponsive to the activating impulse In view of the normal R wave in Lead V_1 and the abnormal QR deflection in Leads V_4 V_5 and V_6 the QS deflection at V_3 may be interpreted as evidence of a central zone of transmural infarction, a conclusion subsequently confirmed at postmortem examination Under such circumstances the ventricular wall merely serves as a window for the transmission of negative cavity potentials to the surface The downstroke of the QS is initiated by negative potentials transmitted to the cavity during activation *of intact portions of the septum and is continued by negative* potentials derived from the posterior and intact portions of the lateral wall of the left ventricle The ascending limb of the QS deflection begins when activation of the outer wall of the left ventricle ceases or when the forces developed by activation of remaining muscle are too small to make the cavity more negative The synchrony of the lower portion of the downstroke apex and upstroke of the QS deflection in Lead V_3 with the S wave in Leads V_1 and V_2 suggests a common *origin The continuation* of the ascending limb above the isoelectric line to an elevated S-T junction may reflect beginning *repolarization of the intact* portions of the septum or right ventricle

The downstroke of the Q wave continues *for an interval of 03 sec in* Lead V_6 and for 04 sec in V_4 and V_5 when it is replaced by an upstroke (Fig 19, c) The transmission of cavity potentials through the anterolateral wall of the left ventricle for 03 sec or longer is abnormal and occurs *when the subendocardial* layer fails to respond to the activating impulse The usual cause is subendocardial infarction Since the cavity is showing increasing negativity during the first 06 sec (as evidenced by the continuing downstroke in V_1 V_2 and V_3) the earlier reversal in the direction of the string in V_4 V_5 and V_6 marks the arrival *of the impulse in responsive* muscle in more

superficial portions of the anterolateral wall The activation of subepicardial muscle is confirmed by the fact that the upstroke crosses the isoelectric line to form a distinct late R wave (Fig 19 d) The time interval of 04 sec to 05 sec elapsing between the onset and peak of the late R wave is abnormally long for activation of the remaining myocardium and may be due to hypertrophy and/or patchy extension of the infarct into the subepicardial layer Thus the lateness of the intrinsicoid deflection and prolongation of the QRS interval are attributable to a lesion of the anterolateral wall and are in part due to a temporary arrest of the impulse in an infarcted subendocardial layer in part to a longer time required for activation of the more superficial layers because of hypertrophy or patchy infarction

The Q waves in Leads V_4 and V_5 are abnormal not only in duration but also in amplitude relative to that of the succeeding R waves This is revealed by increased QR ratios of 100 per cent and 33 per cent respectively in Leads V_4 and V_5 Although the time interval from onset to nadir of the Q wave in V_6 is sufficiently long to suggest subendocardial infarction the QR ratio has only a borderline value of 25 per cent probably due to increase of the R wave as a result of hypertrophy of the more superficial layers

QR patterns in left precordial leads characterized by a downstroke 03 sec or longer in duration and more than 25 per cent of the amplitude of the succeeding R wave are characteristic of subendocardial infarction and are recorded at the margins of a transmural infarct due to the tendency for such lesions to extend much further on their endocardial than on their epicardial surface A rough estimate of the relative thickness of the infarcted subendocardial and living subepicardial layer may be made from (1) the time from onset to nadir of the Q as compared with the time from onset to peak of R and (2) the relative amplitudes of the Q and R waves The equality of the Q and R deflections in Lead V_4 both as to duration and amplitude suggests that the infarct involved approximately the subendocardial half of the subjacent anterolateral wall The progressive decrease in duration and amplitude of the Q in reference to the R wave as the electrode was moved to the V_5 and V_6 positions suggests a progressive thinning of the subendocardial infarct as it extended from the anterior into the lateral wall These deductions are in keeping with the anatomic distribution of the infarct found at autopsy and illustrated in the diagram

The T wave pattern proved fixed in serial tracings The elevated S-T junctions and upright T waves recorded in Leads V_3 and V_4 are not uncommon fixed residues in leads over extensive healed infarcts

Septal infarction without bundle branch block may produce no diagnostic electrocardiographic changes or may be accompanied by a characteristic pattern in right precordial leads whose probable genesis is shown in Fig 20 The pattern is a qrS or a notched QS deflection of 08 to 11 sec duration and differs from the normal rS complex in respect to the direction of the initial deflection The normal r component of an rS deflection is produced in part by activation of the septum from left to right and in part by activation of the free wall of the right ventricle as illustrated in Fig 20 a b The initial downward component of the abnormal qrS complex reflects a right-to-left septal activation (Fig 20 a') reversal of the septal vector can occur as a manifestation of infarction of the left side of the septum or as a result of incomplete left bundle branch block in the absence of gross infarction of the septum Succeeding activation of the free wall of the right ventricle tends to cause positivity of the right precordium indicated by an upstroke which may be sufficient only to produce a notch or may cross the isoelectric line as an r wave (Fig 20 b') The final S wave in both circumstances represents negative potentials transmitted during activation of intact portions of the left ventricle (Fig 20 c) The abnormal qrS diagnostic of infarction of the left side of the septum is accompanied by q waves in leads over the anterior wall of the left ventricle that referable to incomplete left bundle branch block is accompanied by initial slurred (or notched) R waves in all left ventricular leads The abnormal qrS of septal infarction is recorded in leads facing the epicardial surface of the right ventricle primarily V_{3R} V_1 V_2 if the heart is in horizontal position septal infarction of this type may be accompanied by a qrS or notched QS in Lead aV_F

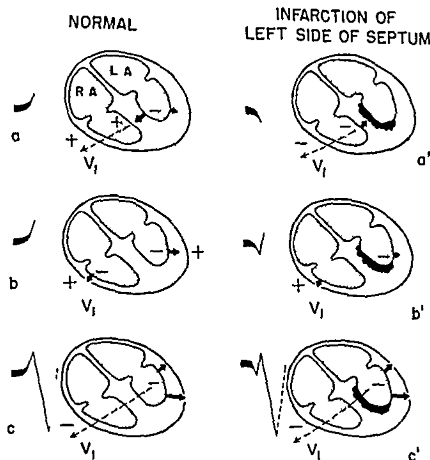


Figure 20

The effects of localized myocardial injury on the QRS-T complex in direct leads from the mammalian ventricle Myocardial injury of variable severity involving the entire thickness of any given portion of ventricular wall or confined to the subepicardial or to the subendocardial layer has been produced in anesthetized dogs and other animals by heat cautery electrocoagulation curettage hypodermic injection of 1/5 M potassium chloride or 95% alcohol local pressure or cold applications The changes in direct epicardial leads found by the various workers were fairly consistent and could be correlated with the distribution of the lesion between the endocardium and epicardium of the underlying and opposite ventricular walls and also with the severity and stage of the injury but not with the noxious agent Furthermore myocardial ischemia and infarction produced by coronary ligation were manifested by changes in direct leads which were comparable to those produced by the foregoing traumatizing agents and which likewise could be correlated with the distribution severity and stage of the ischemic lesion A schematic representation based on the studies of Hellerstein and Katz has been made of the electrocardiographic effects of acute subepicardial injury in Fig 21 and acute subendocardial injury in Fig 22

The cross section of the left ventricle is represented in the illustrations by three cells which correspond respectively to the subendocardial third the middle third and the subepicardial third of the wall A solid border is employed for the membrane of normal cells The thickness of the border reflects the stage of polarization A thick border denotes a resting cell a thin outline signifies a depolarized cell and a partly thick and partly thin boundary indicates a cell in process of activation or repolarization A broken border indicates a moderately damaged cell that responds normally to the activating impulse but requires an abnormally long time for repolarization a solid black contour denotes a severelv injured cell in a constant state of partial depolarization and unresponsive to the activating impulse but not dead The cross sectional diagrams are accompanied by a schematic representation of tracings obtainable by three epicardial leads from the anterior (X) lateral (Y) and posterior (Z) surfaces of the left ventricle before and after injury

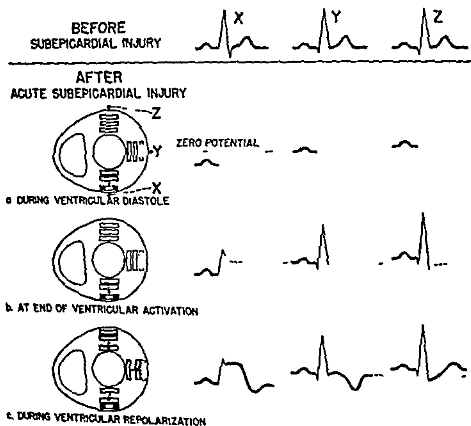


Figure 21

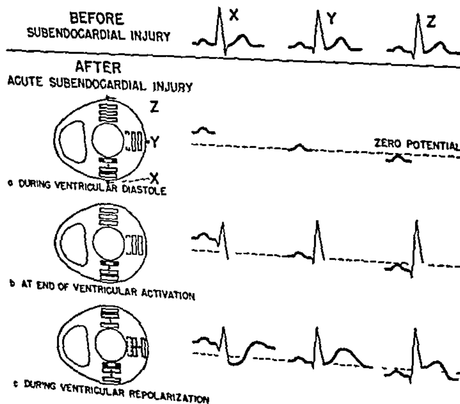


Figure 22

Acute subepicardial injury Fig 21 depicts the effects of severe injury to the subepicardial third of the anterior wall and moderate injury to the subepicardial portion of the lateral wall on the QRS-T patterns obtained by direct leads from the surface of the severely injured anterior wall (X) the moderately damaged lateral wall (Y) and the intact posterior wall (Z) The level of zero potential (true isoelectric line) is represented by a broken line The schematic representation in cludes normal tracings before injury in order to bring out by contrast the abnormalities produced by acute subepicardial injury The registration of the latter is reconstructed in three phase which comprise respectively (a) the portion of the tracing recorded during ventricular diastole (b) the portion recorded up to the end of ventricular activation (c) the complete cycle The accompanying cross sectional diagrams represent the probable electrical status of the ventricles (a) during diastole (b) at the end of activation (c) during the course of repolarization

Continuous tracings taken while severe injury is inflicted on the subepicardial aspect of the anterior wall reveal displacement of the T-P segment below the isoelectric line of zero potential in the anterior epicardial lead (X) elevation of lesser degree in the posterior epicardial lead (Z) and usually no change in the lateral lead This displacement is referable to the development of an electrical bipole at the boundary between the partially depolarized injured subepicardial layer at X and the polarized intact deeper layers and the consequent flow of a current of injury from the latter through extracardiac circuits to the former (the injury current of rest) The downward displacement of the T-P segment recorded at X (Fig 21 a) indicates relative negativity of the injured subepicardial layer the upward displacement of the T-P segment recorded at Z is the result of transmission of positive potential from the deeper uninjured cells through the cavity and opposite wall The lesser magnitude of the displacement at Z than at X is referable to loss of potential in transmission over the greater distance The absence of displacement at Y is merely due to cancelling out of negative and positive potential in a plane perpendicular to the bipole

The horizontal course of the tracing during the inscription of the T-P segment reflects lack of fluctuation in the potential difference between intact and injured cells and constancy of the injury current of rest Unless continuous tracings are taken during infliction of the injury the displacement in the T-P segment is not recognized as primary because of the practice of introducing a compensating current into the galvanometer circuit to exactly counterbalance the sum total of all constant currents picked up from the body including the normal skin current as well as the abnormal cardiac injury current of rest

Study of the portion of the tracing resulting from ventricular activation (Fig 21 b) reveals (1) no change in the QRS deflection recorded at Y indicating no alteration in the response of the lateral wall, (2) a significant reduction in the amplitude of the R wave registered at X attributable to failure of the subepicardial third of the anterior wall to respond to the activating impulse (3) a slight exaggeration in the amplitude of the R wave in Lead Z over the posterior wall secondary to the reduction in oppositely directed electromotive forces arising in the anterior wall Upon completion of ventricular activation the excited subendocardial layer is no longer positive in reference to the injured subepicardial layer and the injury current of rest is no longer present to neutralize the constant and oppositely directed *compensating current* The latter tends to produce a positive displacement as recorded at X and a negative displacement as recorded at Z Hence the S-T junction will appear elevated in reference to the T-P segment in leads from surface covering an area of acute subepicardial injury (X in Fig 21) whereas the S-T junction will be depressed to a lesser degree in leads from the surface of the opposite ventricular wall (Z of Fig 21)

Although one would expect an S-T junction in tracings X and Z at the level of zero potential owing to the unopposed action of a compensating current that had earlier neutralized the injury current of rest examination of the tracings reveals elevation of the S-T junction above this level on the record at X and a lesser degree of depression in the record at Z The direction of the supplementary displacement indicates that the deeper layers of intact myocardium which have just undergone depolarization are more electronegative than the partially depolarized injured subepicardial layer (i e the latter is relatively positive in respect to the former) The electrical bipole at the boundary of damaged and intact muscle at the end of activation (Fig 21 b) is oriented in the

opposite direction from that present at rest (Fig 21 a) and flow of current at the end of activation (injury current of activity) is opposite from that during diastole Continuous tracings during the infliction of subepicardial injury have shown that the S-T elevation in overlying leads and the reciprocal depression in opposite leads may be due to a combination of the effects of the currents of injury and activity or almost exclusively to one or the other, depending upon the individual case Although determination of the relative contribution of these two currents to the S-T displacement is impossible without continuous tracings at the time of injury this is of no clinical significance The features of clinical importance are (1) the recognition of the presence of acute injury and estimation of its severity based upon the demonstration of abnormal S-T displacement of varying degree in serial tracings increasing with extension of the injured zone decreasing or disappearing with recovery or death of the injured cells (2) the localization of the injured area based upon a comparative study of multiple leads

The full T waves are represented schematically in the final tracings but the accompanying cross section (Fig 21 c) shows the ventricular wall during the course of repolarization to bring out the orientation of the bipoles during inscription of the T wave Although the QRS complex at Y was not altered as a result of the moderate injury to the subepicardial aspect of the lateral wall the T wave was reversed in direction and prolonged in duration On the assumption that the normal positivity of the T wave in such a lead is an expression of centripetal repolarization (Fig 3) negativity of the T wave is presumably a manifestation of centrifugal repolarization (Fig 10) The combination of inversion and prolongation of the T wave indicates lengthening in the duration of the excited state in the subepicardial muscle as the reason for the repolarization of the subendocardial layer in advance of the subepicardial layer This supposition is borne out by the fact that cooling of the epicardial surface produces changes in the T wave comparable to those at Y On the contrary T wave inversion caused by digitalis is accompanied by reduction of the Q T interval suggesting shortening in the duration of the excited state of the subendocardial muscle so that repolarization like activation proceeds in a centrifugal direction This explanation is supported by the inversion and shortening of the T wave in an epicardial lead produced by warming the endocardial surface

A study of the ST-T complex at X in comparison with that at Y reveals the marked differences in position of S T segment and junction as discussed above but a resemblance in T waves when evaluated independently of the S T segments At X there is a terminal inversion of the T wave which would resemble the inverted T wave at Y if there were no S-T elevation to influence the over-all contour The resemblance in T wave patterns is probably referable to graduation of the lesion at X from severe injury in the subepicardial layer to moderate injury in the midzone to undamaged myocardium in the subendocardial layer The T wave inversion resembling that at Y is thus an expression of moderately injured myocardium showing normal activation but delayed repolarization and situated deep to the unresponsive subepicardial layer

Acute subendocardial injury Fig 22 the counterpart of the previous diagram is a schematic representation of (1) the anatomic and physiologic alterations produced by severe injury to the subendocardial portion of the anterior wall moderate injury of the same portion of the lateral wall and preservation of the posterior wall (2) the associated electrocardiographic changes as recorded by direct epicardial leads from points X Y and Z respectively The electrocardiographic changes are reconstructed after the findings of Hellerstein and Katz who obtained continuous tracings during the infliction of either mechanical thermal or chemical injury of varying grades on localized portions of the subendocardial layer of the left ventricle

Continuous tracings reveal that sudden severe injury of the subendocardial portion of the anterior wall is accompanied by an abrupt shift of the T P segment above zero at point X and a lesser depression at point Z The recordings through the two electrodes indicate relative negativity of the partially depolarized subendocardial layer and positivity of the intact superficial myocardium with consequent bipole as shown in Fig 22 a resulting in a current of injury This current is transmitted through extracardiac tissues from X to Z and the underlying fluctuations in potential at any two points on the body are registered by appropriate connections to an electrocardiograph

In order to demonstrate that the T-P *shift is primary* a continuous record must be obtained during infliction of the trauma otherwise the current of injury is counterbalanced by adjustment of the compensatory circuit of the electrocardiograph

Activation of the moderately injured lateral wall is not altered and the QRS at Y (Fig 22 b) is not significantly changed from the control The development of a Q wave at the expense of the R wave at X is abnormal in the absence of serial tracings to demonstrate its appearance the presence of a Q wave 03 sec in duration and more than 25 per cent of the succeeding R is abnormal and reflects a delay in onset of activation of the subjacent wall After conduction of the impulse through the bundle of His its activation of the left side of the septum and intact adjoining myocardium results *in the transmission of negative potentials through the cavity* to be recorded as a Q wave in leads over the as yet unactivated portions of the free wall of the left ventricle The polarity at the epicardial surface continues negative until reversed upon arrival of the impulse in intact subjacent myocardium hence increased duration *and amplitude of the Q wave at the expense* of the R wave reflects severe injury or destruction of the subendocardial layer The R wave in leads over the intact posterior wall (Z) is slightly exaggerated secondly to reduction in oppositely directed forces from partial destruction of the anterior wall

Upon completion of ventricular activation the excited subepicardial layer is no longer positive in respect to the injured subendocardial layer and the injury current of rest is abolished but the *constant and oppositely directed compensating current is still present This produces an S-T* depression as recorded at X and an S-T elevation as recorded at Z (Fig 22 g) From momentary obliteration of the resting injury current one would expect that the S-T junctions would be of zero potential however the actual tracings taken during infliction of injury reveal S-T negativity at X and a lesser positivity at Y The direction of supplementary displacement is an expression of the demonstration that the intact subepicardial myocardium which has just been activated is more electronegative than the partially depolarized injured subendocardial layer (i e the latter is relatively positive in respect to the former) The electrical bipole at the boundary of the damaged and intact muscle at the end of activation (Fig 22 b) is oriented in the opposite direction from that at rest (Fig 22 a) In routine interpretations, the position of the S-T junction is determined in reference to the T-P level as if the latter were isoelectric the findings associated with severe injury confined to the subendocardial portion of the anterior wall are *characterized by S-T depression in* leads facing the epicardial surface of the anterior wall (X) and a lesser degree of elevation in leads facing the opposite wall The two component factors in this *displacement have been discussed above*

The full T waves are represented schematically in the final tracings but the accompanying cross section (Fig 22 c) shows the ventricular wall during the course of repolarization to bring out the orientation of the bipoles during inscription of the T wave The injury at Y was not severe enough to modify activation of the lateral wall as shown by lack of change in the QRS but did prolong repolarization as indicated by lengthening of the T wave and hence the Q-T interval The maintenance of positivity during inscription *of the T wave indicated that repolarization was proceeding in* the normal centripetal direction the prolongation of the T wave was presumably referable to delayed recovery of the moderately injured subendocardial layer If the S-T displacement at X is disregarded the late upright T wave resembles that at Y and is presumably referable to a centripetal repolarization that is prolonged in a moderately injured midzone bordering on the severely traumatized subendocardial layer

D. DISORDERS OF RATE AND RHYTHM

INTRODUCTION

I ANATOMIC BASIS Histologically distinctive muscle fibers together with terminal twigs from the vagus and sympathetic nerves are arranged anatomically into two principal nodes (1) the sinoauricular node of Keith and Flack (S-A or sinus node) composed of a head 5 mm in diameter located on the superior surface of the right atrium adjacent to the orifice of the superior vena cava and a tail 20 mm long extending in a dorsocaudad direction towards the orifice of the inferior cava (2) the auriculoventricular (A-V) node of Tawara which along with the anatomically continuous auriculoventricular bundle of His forms a compact band, 3 mm in diameter connecting auricles and ventricles, and divisible, for descriptive purposes into the following three portions (a) upper or atrial part 4 mm long and located on the right side of the posterior aspect of the interatrial septum above the attachment of the medial leaflet of the tricuspid valve and anterior to the coronary sinus (b) middle portion, passing through the connective tissue at the atrioventricular junction (c) lower or ventricular portion continuing in an anterior and caudad direction for a distance of 10 mm to the membranous portion of the septum where it divides into two branches

The right branch lies beneath the endocardium of the right side of the septum and can be traced for a distance of 25 to 50 mm where it divides into the Purkinje network the left branch passes through the membranous portion of the septum to reach the endocardial surface below the commissure of the right and posterior aortic cusps splits into an anterior and posterior division then immediately breaks up into a complex Purkinje network The fibers that make up the Purkinje plexus are histologically distinctive but intimately associated with the remainder of the myocardium They fan out beneath the endocardial surfaces of both ventricles and extend towards the epicardium in an almost perpendicular direction on the left and a more oblique direction on the right Small collections of specialized muscle cells are scattered over both atria but do not appear to form a continuous network like the Purkinje plexus of the ventricles

II PHYSIOLOGIC BASIS Normal cardiac activation is accomplished through the following steps (1) generation of an impulse within the S-A node (2) discharge into the adjoining right atrium, (3) radial spread through the right and left atria (4) transmission through the A-V node bundle branches and Purkinje network to the myocardium of the septum and both ventricles (5) centrifugal spread away from the subendocardial layer of muscle towards the center of the septum and to the epicardium of the free walls of both ventricles

A LOCATION OF PACEMAKER All myocardial cells possess the inherent properties of generating impulses and conducting them to contiguous cells The focus from which the

activating impulse arises is designated as the pacemaker The S-A node has the greatest inherent capacity for impulse formation and, therefore, serves as the normal pacemaker for the heart All pacemakers outside of the S-A node are designated as ectopic and are classified according to location as auricular, A-V nodal or ventricular These groups may be subdivided into right or left auricular, upper, middle or lower nodal, septal right or left ventricular, and further, into basal or apical ventricular

B NUMBER OF PACEMAKERS The entire heart is customarily activated from a single pacemaker This is normally located in the S-A node, but may be ectopic, if the S-A node is depressed or if an outside focus is sufficiently hyperirritable to generate impulses more rapidly than the S-A node and thereby usurp control of the heart Many records show evidence of more than one pacemaker and may be classified into two groups (1) concurrent but separate pacemakers for auricles and ventricles, encountered in complete A-V block and in interference dissociation, (2) consecutive pacemakers for the entire heart The latter may consist of (a) the S-A node and one or more ectopic foci, with a resultant sinus mechanism interrupted by ectopic beats coming singly or in short runs, (b) two or more ectopic foci which consecutively usurp control of the entire heart

C MECHANISM OF ORIGIN OF ECTOPIC BEATS AND RHYTHMS The two major types, as judged electrocardiographically will receive separate consideration

1 Escape is a physiologic mechanism that comes into play when the S-A node is depressed and serves to protect against prolonged cardiac standstill Escape occurs because of the inherent property of any part of the heart to take over as pacemaker when the S-A node ceases to function Isolated escaped beats are characterized by (a) an antecedent pause, distinctly longer than the customary diastolic interval (b) a configuration indicative of an ectopic origin (generally A-V nodal occasionally ventricular or auricular) An escaped rhythm represents a series of escaped beats

2 Accelerated impulse release from an ectopic focus Premature beats may occur singly or in runs and are characterized by (a) appearance in advance of the next anticipated beat (b) configuration indicative of an ectopic origin (most commonly ventricular but also auricular or A-V nodal) Although readily recognized electrocardiographically, the etiology is often indeterminate and the mechanism of production is speculative It is probable that there is no single mechanism common to all cases with premature systoles or with the closely related ectopic tachycardias The findings in some cases are consistent with one or more hyperirritable ectopic foci, the findings in others are better explained by the theory of re-entry

a Hyperirritable ectopic focus Application of an electric shock mechanical or chemical irritant to nonrefractory myocardium provokes the release of one or more impulses which spread radially from the irritated focus to activate the heart A momentary stimulus generally elicits a single ectopic beat but may initiate a paroxysm of tachycardia, provided that (1) the myocardium is predisposed by vagal stimulation ischemia or poisoning (2) the stimulus is applied just after the end of the refractory period Repetitive stimuli may produce ectopic tachycardia flutter or fibrillation depending upon the frequency and the state of the myocardium

The close relationship of premature systoles ectopic tachycardia flutter and fibrillation is exemplified by the similarity of experimental production One or more hyperirritable foci constitute the presumptive background for many but not all clinical cases with premature systoles ectopic tachycardia flutter or fibrillation

The hyperirritability may be conditioned physiologically by way of the autonomic nervous system through reflexes from distended abdominal viscera or from irritated somatic sites or through cerebral discharges accompanying cerebral disease emotion and occasionally even cerebration it may be conditioned chemically as a result of alkalosis or exogenous agents such as tobacco alcohol caffeine etc Premature systoles or ectopic tachycardias may occur under such conditioning influences in

persons with apparently normal hearts They also may arise from hyperirritable foci secondary to structural disease such as inflammation ischemia etc Premature systoles, referable to ischemia or structural disease may appear on exercise whereas those in normals are generally dissipated by exercise because of the acceleration in the sinus mechanism

b [illegible] time interval between the [illegible] ith coupled rhythm or fre- [illegible] was instrumental in the genesis of the premature beat This has been explained by re-entry a theory based upon the premise of significant differences in the length of the refractory period in contiguous portions of the myocardium The existence of a localized area of refractory myocardium in the path of the advancing impulse of the normal cycle is postulated If this localized area of myocardium is still completely refractory when first reached the impulse will by pass it if it recovers soon the impulse may subsequently enter it from another direction If the myocardium in the localized zone is partially refractory the impulse may persist unduly long in this area In either event, the excited state may persist in this localized area after recovery of the surrounding myocardium from the effects of activation

The excited myocardium becomes a focus for radial redistribution of the impulse to the rest of the heart manifested by the inscription of an ectopic deflection always coming at a fixed interval after the preceding normal cycle If a portion of the myocardium surrounding the ectopic focus has not recovered at the time of emergence of the impulse it may advance in a single instead of a radial, direction Because of the approximately circular shape of the heart a unidirectional impulse may be either clockwise or counterclockwise Propagation will be expected as long as the myocardium immediately in advance has regained responsiveness If the impulse is able to continue it will return to the original focus to complete a circuit Repeated movement of the impulse around the same circuit may constitute one mechanism for ectopic tachycardia and flutter

D CONDUCTION OF IMPULSES FROM AURICLES TO VENTRICLES may be abnormal in the sense of (1) retardation or block (2) acceleration

1 Retardation or block. Passage of impulses from auricles to ventricles by way of the A-V node and bundle branches is governed not only by the anatomic and physiologic integrity of the junctional conducting tissue but also by the tone of the innervating vagal fibers Thus A-V block may be classified according to pathogenesis into three overlapping groups

a Anatomic block Ultimate classification into this category is based upon histologic evidence of a lesion invading or compressing the A-V node and/or both [illegible] branches in some cases with complete A-V [illegible] [illegible] ... e obtained ... bundle of ... [illegible] e en though meticulous effort is [illegible] continuity of the junctional tissues through serial sections Thus in spite of dense patches of destruction normal A-V conduction may have been maintained during life as the result of the preservation of a few neuromuscular strands connecting auricles and ventricles on the other hand in patients with lesions much less [illegible] ing ... ive been present during as ... histologically such [illegible] like digitalis and quinidine and particularly neurogenic depression from vagal hyperirritability

Clinical classification of A-V block into the anatomic group depends upon the recognition of an etiologic agent capable of damaging the bundle of His but does not stop at this point since a complete study must include an evaluation of the contributory effects of physiologic factors such as ischemia, digitalis e c and neurogenic depression

from vagal hyperirritability, to be discussed below. The anatomic lesions which may form the basis for A-V block may be classified as follows:

1) Congenital defects are statistically important causes of complete A-V block, but are relatively rare cases of partial A-V block. Congenital A-V block may be secondary to patent interventricular septum, but occurs in only a minority of the cases with this defect, because of the fact that the aperture is usually anterior to the bundle of His. Congenital A-V block may date from the earliest clinical signs of cardiac activity in utero and has been diagnosed antenatally from a slow fetal heart rate or it may not appear until years after birth. Late development in patients with patent interventricular septum has been ascribed to eventual degenerative changes from incessant mechanical impingement of blood passing through the defect. The diagnosis of congenital A-V block is made readily in children and less easily in adults on the basis of the following criteria: (1) electrocardiographic evidence of A-V block, usually complete, (2) history of a slow pulse and/or syncopal attacks from an early age, (3) physical signs of a congenital lesion, usually with patent interventricular septum, (4) exclusion of other causes.

2) Myocarditis. Acute infections constitute a frequent background for partial A-V block, but a relatively uncommon cause of complete A-V block. The acute infections in which A-V block is a classical complication include diphtheria, rheumatic fever, and other hemolytic streptococcal infections, such as scarlet fever and septic sore throat. Although the presence of A-V block in febrile patients is often taken as presumptive evidence of acute rheumatic fever, recent studies have demonstrated A-V conduction defects in many other infections, particularly virus diseases, such as influenza, mumps, poliomyelitis, and even German measles.

   Indirect electrocardiographic evidence of an inflammatory etiology for the A-V block is present in some cases in the form of S-T patterns referable to subepicardial injury, arising in association with pericarditis. The presence of an inflammatory or toxic lesion in the junctional tissue may be inferred when high grade block develops during the height of the infection, especially when bacteremia is present or when the agent is known to produce an exotoxin, as in diphtheria.

   The mere fact that infection is present does not imply that an inflammatory process in the A-V node is responsible for the conduction defect. The frequent appearance of the conduction defect during convalescence or at a time when allergic reactions are prone to occur suggests that hypersensitivity may constitute the background in many cases. Such a concept is supported by the experimental production of A-V block in rabbits by anaphylactic shock.

   Vagal hyperirritability is an important contributory factor in conduction defects associated with infection, particularly in those developing during convalescence and in those accompanying acute rheumatic fever, as shown by the fact that atropine reduces the P-R interval to normal in some cases and shortens it significantly in many many others. Underlying hyperthyroidism appears to render the A-V node susceptible to functional impairment during intercurrent streptococcal infection and has been dignified by the description of a triad, consisting of exophthalmic goiter, rheumatic fever, and A-V block.

   The conduction defects associated with acute infection are usually transitory and almost invariably disappear after recovery. An occasional exception may occur in diphtheria, which has been suspected as a cause of permanent complete block, and in recurrent rheumatic fever, ending in inflammatory fibrosis. Granulomas invading or compressing the bundle of His, such as syphilitic gumma, are rare causes of permanent A-V block.

3) Acute infarction. A-V block may occur as a complication of extensive posterior infarction, owing to the fact that the A-V node receives its nutrition from branches

of the vessels that supply the posterior aspect of the ventricles namely, the right coronary artery in 90% and the left circumflex in 10% of the cases however A-V block is an uncommon complication of posterior infarction owing to the fact that the right coronary branch that customarily supplies the A-V node and bundle comes off proximal to the usual sites of occlusion When A-V block is produced the cause is revealed by the changes in QRS-T pattern diagnostic of recent posterior infarction If the patient survives normal A-V conduction is often restored

4) Degenerative changes including those secondary to coronary disease are responsible for the majority of cases of complete A-V block and high grade partial block The histologic lesion generally consists in fibrosis, occasionally calcification rarely fatty degeneration Calcification of the septum in the vicinity of the bundle of His has been reported as a manifestation of an extension of a calcified aortic stenosis or mitral annulus and a complication of Paget's disease and might be suspected as a cause of complete A-V block under these circumstances Aneurysm of the sinus of Valsalva has been reported to cause complete A-V block by compression of the bundle of His

5) Neoplasm may produce A-V block by invasion or compression of the bundle of His A presumptive diagnosis is possible when A-V block develops in a patient with a disseminated neoplasm such as leukemia metastatic sarcoma, or carcinoma

b Physiologic and/or [illegible]
re[illegible]
st[illegible] [illegible] pharmacologic depressants such as toxic doses of quinidine and digitalis Cardiac glycosides are very frequent causes and produce block in part by a direct myocardial effect in part by vagal stimulation The reported association of *complete A-V block with* myxedema and *recovery under* thyroid is presumably another example of physiologic block The predisposing influence of hyperthyroidism has been mentioned above

c [illegible] hyperirritability As em- [illegible] A-V node is an aggrava- [illegible] major cause of block in many patients with organic lesions Prolongation in the P-R interval has been observed in a number of healthy young adults as a manifestation of *vagotonia* whereas partial A-V block and rarely complete block have been produced by mechanical stimulation of the carotid sinus or by vagal reflexes of esophageal origin in *persons* with no clinical evidence of an organic lesion of the A V *node* The vagal origin of the conduction defect has been confirmed by the improvement or abolition after atropine

2 *Acceleration* An impulse of sinoauricular origin may elicit a P wave and a QRS complex after an interval less than 12 *sec* because of either (a) accelerated transmission through the A V node without alteration in QRS contour (b) transmis*sion through* an anomalous pathway in which event the QRS is aberrant in form The customary designation of the latter by proper names the Wolff-Parkinson-White (W-P-W) *syndrome* rather than descriptive terminology arose pending the outcome of a *controversy* over its mechanism and gives credit to the fact that the detailed account by *Wolff* Parkinson and White in 1930 was largely respons*ible for* directing attention to this disorder of conduction notwithstanding previous reports of isolated cases Several theories as *to* mechanism have been proposed but that based on anomalous ventricular *excitation* through the bundle of Kent has been adopted *because it* best explains the anato*mic* and physiologic findings

a Anatomic evidence The bundle of Kent extending as a *neuromuscular* bridge from the auricle across the atrioventricular groove to the epicardial aspect of the ventricle is consistently present in certain lower animals and occurs as an anomaly in man demonstrated in carefully studied autop*sied* cases of the W-P-W syndrome

b Physiologic evidence Transmission of impulses through the bundle of Kent was demonstrated in the beating mammalian heart after severance of all other connections between atria and ventricles and occurs in advance of transmission through the A-V node in various birds and in calves The latter is in accordance with the *electrocardiographic findings* which in summary consist of (1) P waves of sinoauricular origin, (2) shortening of the P-R interval below 12 sec as a result of an abnormally early onset of the ventricular complex (3) prolongation of the QRS duration at the expense of the P-R interval, so that, when the syndrome is intermittent, the time from the onset of the P to the S-T junction is the same in anomalous as in normal cycles, (4) a *fusion QRS* consisting of a thick-imaged (slurred or notched) premature component and a thin-imaged steep final component (5) delay in onset of the intrinsicoid deflection in measurements made from the beginning of the QRS, but preservation of its normal precipitous descent, and (6) tendency to paroxysms of tachycardia

c *Reduplication of all of the electrocardiographic features* of W-P-W syndrome has been accomplished in normal animals by pickup of the impulse after emergence from the sinus node and transmission to the ventricular epicardium at any predetermined time prior to its conduction through the bundle of His Electrocardiograms simulating those in humans with the W-P-W syndrome were obtained in normal cats and dogs prepared by the attachment of small silver electrodes to the surface of the right atrium and ventricle and by connection of the former to the input the latter to the output of an amplifier The auricular action current responsible for the P wave was picked up by the electrode on the surface of the right atrium amplified and transmitted to the surface of the ventricle at any predetermined time prior to the arrival of the impulse at the bundle of His

In tracings obtained by this procedure the P-R segment was shortened or obliterated *by the premature onset of the ventricular deflection the initial portion of the QRS* was coarsely slurred or notched and the remainder of the complex resembled that in the control tracings taken without the short circuit The premature slurred component of the QRS was inscribed by spread of the short-circuited action current from its point of application on the epicardial surface into the adjoining ventricular myocardium, its replacement by the normal final component evidently resulted from extinction of this action current by the arrival of the normal impulse distributed through the ramifications of the bundle of His and followed by activation of the remainder of the ventricles in the usual manner The longer the interval between the arrival of the short-circuited action current and the normal impulse the more of the ventricles activated in the aberrant fashion and the greater the distortion of the ventricular complex The QRS deflections obtained by conduction of the short-circuited action currents to the right ventricle were opposite in direction from those recorded by transmission to the left ventricle A paroxysm of supraventricular tachycardia was produced by picking up the ventricular action current during the registration of the QRS and returning it to the auricle for retransmission through the atrioventricular node

D PRINCIPLES OF CLASSIFICATION From the electrocardiographic standpoint the rhythm may be classified in accordance with the location of the pacemaker into four major groups I Exclusive or predominant sinus pacemaker, II A-V nodal pacemaker, III Ectopic auricular focus, IV Ventricular focus Each of the last three categories is divisible into two principal subgroups depending upon whether the ectopic focus merely supplements or actually replaces the sinus pacemaker Further subdivisions in the classification will be brought out *through a systematic presentation* which differs from that in Section B in the manner of organization rather than the nature of the contents The objective in Section B was to present a method of analysis of the tracing for the purpose of determining cardiac rhythm whereas the objective of Section D is to present an organized description of the electrocardiographic features of each disorder of rate and rhythm For example it was necessary to take up the various forms of nodal rhythm in five separate portions of Section B all of these data are collected and organized into one portion of Section D

DISORDERS OF RATE AND RHYTHM — CLASSIFICATION AND ELECTROCARDIOGRAPHIC FEATURES

I EXCLUSIVE OR PREDOMINANT SINUS RHYTHM

A REGULAR (UNCOMPLICATED) SINUS RHYTHM is present when the S-A node maintains exclusive control over the heart by regular discharge of impulses that are transmitted in a uniform manner to reach the ventricles within the normal time limits

1 Electrocardiographic features

a Regular spacing of the P waves and QRS complexes Sinus rhythms are considered regular in the presence of slight fluctuations in cycle length not to exceed 12 sec Sinus mechanisms which show variations of more than 12 sec between the longest and shortest cycles fall into the category of sinus arrhythmia

b Constant P-R interval falling within the normal limits The influence of heart rate and age are discussed on page 17

c Registration of inverted P waves in Lead aV_R and upright in aV_I

d Uniformity in the shape of the P waves and QRS T complexes in any given lead Exceptions may occur in a precordial lead near the transitional zone or in Leads aV_L and aV_F as a result of respiratory shifting in cardiac position The respiratory origin may be suspected when the rhythmic alterations in contour recur every 3 to 4 sec and may be confirmed by the absence of fluctuations in contour in a second tracing taken while breathing is suspended

2 Classification according to rate In the resting patient with regular sinus rhythm the rate should fall between 60 and 100 beats per minute The following variants are noted

a Sinus bradycardia is characterized by a sinus mechanism with rate below 60 The rate is usually between 40 and 60 but rarely may fall below 40 and is often, although not invariably accompanied by sinus arrhythmia Sinus bradycardia may occur in persons with normal hearts particularly in athletes may be an expression of excessive vagal tone as in increased intracranial pressure ocular pressure or carotid sinus pressure or may be the result of certain intoxications as jaundice digitalis quinidine and tobacco Before arriving at a diagnosis 2 1 A-V block must be excluded by a deliberate search for blocked P waves as discussed on page 17 A 2 1 sino-auricular block is indistinguishable from sinus bradycardia until a sudden change in ratio occurs at which time an abrupt doubling of ventricular rate will be noted if the ratio becomes 1 1 or an irregularity develops if there is a change to a 3 2 or lower ratio

b Sinus tachycardia is characterized by a sinus mechanism with a rate above 100 The rate is usually between 100 and 140 but may be as high as 180 Sinus tachycardia occurs physiologically in response to exercise heavy meals and emotional stimuli, and in association with fever thyrotoxicosis hemorrhage, shock etc in the absence as well as in the presence of cardiac disease The differentiation between sinus and ectopic tachycardias is given on page 2[illegible]

B SINUS [illegible]
form [illegible] impulse
vari[illegible] exceeding 12 sec It is a common finding parti[illegible] hil-
dren and in adults over 50 and results from [illegible] ar-
rhythmia is a physiologic mechanism it m[illegible]
carotid sinus and often accompanies cardia[illegible] example in children it may be
found in the presence of acute rheumatic carditis in elderly persons its association with

advanced coronary disease is more frequent than would be expected from chance Thus the presence of sinus arrhythmia should prompt a search for clinical and electrocardiographic evidence of cardiac disease and a test for carotid sinus hypersensitivity before a final interpretation is made If these prove negative, sinus arrhythmia should be regarded as a normal variant Sinus arrhythmia may be classified into two main subgroups depending upon whether the point of exit from the S-A node and spread through the atria are constant (as in the uncomplicated form) or variable (as in wandering pacemaker)

1 Uncomplicated sinus arrhythmia is present when cycle lengths differ by more than 12 sec while P waves and QRS complexes are uniform in contour and in relationship in any given lead Two types may be recognized

a Phasic type the common variety, is manifested by rhythmic waxing and waning in the duration of the cardiac cycl ... nd lengthening of the T-P segment, related tc ... The shortest and longest cycles in a given le ... of sinus arrhythmia is merely an exaggeration of the normal tendency for acceleration towards the end of inspiration and slowing towards the end of expiration The P waves and QRS-T complexes are characteristically uniform in configuration in any given lead but may show slight variations in contour, especially in aV_L, aV_F, and transitional precordial leads, as a result of respiratory shifting in cardiac position The P-R interval is constant

b Nonphasic type is characterized by irregular variations in cycle length in the presence of P waves of uniform contour and P-R intervals of constant duration Conversion to the phasic variety is generally demonstrable by repetition of the tracing during deep breathing

2 Sinus arrhythmia with shifting pacemaker Wandering of the pacemaker within the S-A node or transitory shift to the A-V node are fairly common in marked sinus arrhythmia and are easily recognized if attention is directed to the relationship of P and QRS deflections in each cycle

a Sinus arrhythmia with wandering sinus pacemaker resembles the uncomplicated phasic or nonphasic variety in respect to spacing of the P waves but is differentiated by the presence of distinctive variations in the contour of the P waves accompanied by slight differences in the duration of the P-R interval The P waves tend to be taller and more sharply peaked in the shorter cycles lower and more rounded in the longer cycles Shifting in the point of exit of the impulse from the 2 5 cm long S-A node alters the vector associated with activation of the auricles, thereby accounting for the change in contour of the P wave, and also modifies the time of arrival of the impulse at the A-V node and thus the duration of the P-R interval

b Sinus arrhythmia with nodal escape and interference In the waning phase of marked *sinus arrhythmia the diastolic pauses* may be so long that an ectopic center in the auriculoventricular (A-V) node discharges an impulse into the ventricles before the sinus impulse arrives This is known as nodal escape and is recognized by a preceding pause distinctly longer than the customary T-P interval at the end of which there is a QRS-T complex similar to those of sinus origin but independent of the P wave

If a sinus impulse is released before the nodal impulse can be conducted backward into the auricles a P wave similar to those of sinus origin, should be visible either just ahead of the QRS (at a P-R interval less than 12 sec) or following the QRS or may be obscured by superimposition upon the QRS * *During such a cycle the ventricles* are controlled by the A-V node the auricles by the sinus node, a condition known as interference

*If the impulse from the A-V node is conducted backward and activates the auricles before an impulse is released from the sinus node the escaped QRS complex is followed by a retrograde P wave which differs in shape from P waves of sinus origin

If the sinus node recovers in time to gain control over the entire heart at the next cycle the previous beat is referred to as an escaped nodal beat with isolated interference On the other hand the A-V node may control the ventricles for several successive beats while the sinus node controls the auricles resulting in an escaped nodal rhythm with interference dissociation Single or multiple escaped A-V nodal beats represent a physiologic response to depression of the sinus node and are normal manifestations of the irritability and rhythmicity inherent in cardiac muscle

C SINUS RHYTHM COMPLICATED BY TRANSITORY CARDIAC STANDSTILL is manifested by an underlying regular sinus mechanism that is interrupted by pauses of two cycle lengths or longer during which there is no evidence of either auricular or ventricular activity The fact that the rhythm preceding and following the pauses is either perfectly regular or nearly so distinguishes this disorder from marked sinus arrhythmia in which there are also considerable variations in cycle length Before arriving at a diagnosis, blocked auricular premature beats (page 106) and partial A-V block (page 96) must be excluded by deliberate search for isolated P waves throughout the pause and particularly on the T wave of the last cycle

The electrocardiographic features of transitory cardiac standstill depend both on the mechanism and mode of termination Transitory cardiac standstill in an otherwise regular sinus rhythm may be classified according to mechanism into two types, namely sinus arrest representing a transitory cessation of impulse formation in the S-A node, and sinoauricular block constituting an interruption in transmission to the right atrium Both forms are usually the result of excessive vagal stimulation but are occasionally traceable to a lesion of the S A node * Factors that operate through the vagal pathways include pharmacologic stimulants such as digitalis mechanical irritation of either the medullary center (e g from increased intracranial pressure) the carotid sinus or rarely the peripheral portion of the nerve or reflex irritation from stimuli arising in the afferent endings in the esophagus or stomach factors acting directly on the S-A node include pharmacologic depressants such as quinidine and local lesions such as inflammation ischemia infarction and hemorrhage Cardiac standstill from vagal stimulation may be counteracted by pharmacologic antagonists such as atropine The electrical activity of the sinus node is recordable through direct leads but not through precordial or limb leads therefore, block in the formation or release of impulses from the sinus node is recognized from the abnormal suspension of both auricular and ventricular activity Sinoauricular block and sinus arrest are differentiated from one another by the mathematical relationship of the duration of the cycle embracing the pause to the customary cycle length

1 S [illegible] ...uses during which there is com- [illegible] with consequent prolongation [illegible] ...ately double (occasionally treble or quadruple) that of the underlying sinus rhythm These pauses may occur at irregular intervals or regularly after every second third or fourth impulse In the event that every other impulse generated in the sinus node fails to reach the auricles the resultant pattern is indistinguishable from marked sinus bradycardia until a change in ratio of sinus beats to auricular response occurs Restoration of a 1 1 ratio causes an abrupt doubling of both the auricular and ventricular rates, substitution of a 3 2 ratio causes coupling of the P-QRS-T complexes with pauses of suspended auricular and ventricular activity, lasting two cycle lengths Sinoauricular block may be further classified into two types, depending upon the underlying sinus rhythm

[illegible]

a Sinoauricular block with an otherwise regular sinus rhythm is the classical variety The P-P interval is uniform except for the cycle embracing the pause, which is an exact multiple (usually 2X occasionally 3X or 4X) of the customary cycle length

b Sinoauricular block with a slight, but progressive, lengthening of the P-P interval preceding the pause is a variant, to be distinguished from sinus arrhythmia, in which the progressive changes in cycle length are more marked The slight lengthening of the P-P interval is presumably an expression of increasing time required for passage of the impulse through the fatiguing bridge between S-A node and atrium whereas the pause represents a transitory complete interruption of conductivity Proof of the foregoing hypothesis has not been elicited in man because of the fact that the potential variations of the S-A node are too small to be registered in the usual leads but has been obtained in the dogfish by means of a direct lead from the S-A node The findings were thus analogous to the Wenckebach phenomenon in partial A-V block

The progressive lengthening in human S-A block may begin with the first cycle after the pause or not until after the second cycle In the latter event the first P-P interval after the pause is shorter than the second, because of the fact that the rested conducting tissue transmits the first impulse more rapidly from S-A node to atrium to give a relatively early P wave, the second more slowly to give a relatively late P wave

2 Sinus arrest The episode of complete suspension of both auricular and ventricular activity is longer than that in S-A block and is not a multiple of the customary cycle length Sinus arrest, as a rule, is a manifestation of the carotid sinus syndrome and may occur spontaneously or may be induced by pressure upon either of the carotid sinus bodies With protracted sinus arrest, ventricular control is taken over by the A-V node or an idioventricular pacemaker and the underlying auricular standstill is recognized by the absence of sinus P waves from all leads including precordial leads at the right sternal border and when feasible esophageal leads *

3 Mode of termination of the pause may take one of three forms

a Resumption of sinus activity The beat terminating the pause is characterized by a P wave P-R interval and QRS-T complex similar to that of other cycles

b Nodal escape The pause is terminated by a QRS-T complex similar to that of other cycles accompanied by either a retrograde P wave (upright in aV_R inverted in aV_F) or an independent sinus P wave This P wave is identified as of sinus origin by a contour similar to that of the P waves of the underlying sinus rhythm and is judged independent of the ventricular deflection from its position either just ahead of the QRS (at a P-R interval less than 12 sec) or superimposed upon the QRS or following it

c Ventricular escape The pause is terminated by a bizarre QRS-T complex quite different from that occurring in response to supraventricular impulses and may be accompanied by an independent sinus or retrograde P wave similar to those seen in nodal escape

D SINUS RHYTHM COMPLICATED BY ABNORMAL AURICULOVENTRICULAR CONDUCTION The abnormality may take one of two directions namely (1) retardation or block (2) acceleration

1 Retardation or block The electrocardiographic findings yield conclusive evidence as to the presence or absence of A-V block and establish the degree of block but do not reveal the etiology except in cases with patterns pathognomonic of infarction The etiologic

*In sinus arrest with A V nodal or ventricular pacemaker auricular standstill may be prevented by retrograde excitation however tracings of this type are classified as nodal (or idioventricular) rhythm with retrograde auricular activation rather than sinus arrest

diagnosis ordinarily rests on clinical criteria as discussed above, but is sometimes obscure until autopsy

Errors in electrocardiographic interpretation are sufficiently common to necessitate re emphasis of ... he nature of the P ... ave With prolongation ... of the preceding cycle it is usually identifiable as a notch but may be perfectly fused with the summit of the T wave Under these circumstances the P wave may go unrecognized and an erroneous diagnosis of middle nodal rhythm or auricular standstill may be made When no separate P waves are discernible the possibility of superimposition on the T waves should be considered particularly when the latter are pointed and should be investigated by taking extra leads in which the P waves are characteristically diphasic and high in voltage (such as HV_1 or esophageal leads behind the atrium) A 2 1 A-V block may be missed through failure to recognize the blocked set of P waves, as a result of superimposition on the T of the preceding cycle This source of error may usually be avoided by careful inspection of all leads since there is usually sufficient variability in A V conduction so that P and T are not perfectly fused in every cycle of all leads With the aid of dividers set at one-half the R-R interval and applied with one point on the summit of a well-defined P wave the blocked P wave may often be detected by a search of the portion of the tracing where the other point is expected and its temporal position in the auricular cycle established by demonstrating that it falls midway between two well-defined P waves

When the relationship of P and QRS-T is variable the auricular and ventricular rates should be measured and the entire tracing should be studied with dividers first, to differentiate between interference dissociation and A-V block and second, to ascertain the degree of block Although the former objective may be more understandable to beginners after familiarity with interference dissociation, described on page 109 and A-V block page 94 consideration of their major differences is essential to the avoidance of errors in the diagnosis of A-V block

Partial and complete A-V block result from failure of the ventricles to respond to some or all impulses of auricular origin, and as a consequence the ventricular rate is slower than the auricular On the other hand interference dissociation is an expression of a second more active pacemaker in the A-V node or ventricle, that usurps ventricular control from the sinus node intermittently or continuously and causes the ventricles to beat either at a more rapid rate than the auricles or at approximately the same rate but slightly in advance of the arrival of the sinoauricular impulse hence the failure of a ventricular response to a sinoauricular impulse is always explainable by QRS and P relations that indicate arrival of this impulse while the ventricles are refractory If the P wave is sufficiently late to indicate arrival of the sinoauricular impulse after the ventricles have recovered from the refractory state a QRS response should be demonstrable and will indicate that this disorder is interference dissociation rather than A-V block The differentiation of partial and complete A-V block is considered in the respective descriptions below It is customary to classify A-V block into three degrees arranged in order of increasing severity

a Delayed A V conduction (first degree block) is manifested by P R prolongation Since the interval from the onset of the P wave to onset of the QRS is a measure of the time required for the spread of the impulse through the atria and for its passage from auricles to ventricles the P R interval may be lengthened by defects in conduction through the atria as well as through the junctional tissue The former are recognized by prolongation in the duration of the P wave beyond .11 sec the latter by lengthening in the P R segment

1) Criteria for diagnosis consist in

a) P-P interval exceeding the upper limits of normal (page 17) as a result of lengthening in the P Q segment

b) QRS response to every P wave without any dropped beats This separates first and second degree block Furthermore, the P-R interval is usually constant in the former and variable in the latter

2) Evaluation of the role of vagal hyperirritability should be carried out, whenever possible, by repetition of the tracing after change from a recumbent to erect posture, after exercise or a therapeutic dose of atropine

3) Classification according to severity may be made into the following three groups

a) Slight prolongation, up to 28 sec , has been found in a number of healthy persons, as well as in association with disease known to affect junctional tissue The common denominator in both groups is vagal hyperirritability Because of its demonstration in healthy persons, P-R prolongation should not be construed as definite evidence of a myocardial lesion, when present as an isolated finding

b) Moderate to marked prolongation, represented by P-R interval between 28 and 50 sec , usually has an organic background, but has been observed in apparently healthy persons Vagal hyperirritability is often an important contributory factor in the former and is solely responsible in the latter

c) Extreme prolongation is a rare finding but may reach limits of 80 to 88 sec

b Partial A-V block (second degree block) The ventricles are still under auricular control, but conduction is sufficiently impaired that some impulses fail to pass through the A-V node This results in dropped ventricular beats and makes the ventricular rate slower than the auricular

In the analysis of complex tracings the influence of an impulse that has penetrated the A-V node on the ability of the junctional tissues to conduct a second impulse must be borne in mind After the entrance of an impulse, the junctional tissues pass physiologically through the following stages in respect to their capacity to transmit a second impulse (1) an immediate state of absolute refractoriness, followed by successive stages of (2) partial refractoriness, (3) supernormal conductivity (4) normal conductivity When the junctional tissues are injured or depressed the first two stages are prolonged A second impulse will be blocked if it reaches the A -V node during the stage of absolute refractoriness or will be delayed in its passage, but eventually transmitted, after a prolonged P-R interval if it arrives during the second stage The supernormal phase becomes of clinical significance only when the junctional tissues are markedly depressed, in which event impulses arriving during the supernormal phase are less delayed in transmission than those arriving later Thus, when one impulse arrives after the supernormal phase and the next during it, paradoxical shortening of the P-R interval may be observed When the junctional tissues are able to transmit only those impulses arriving during the supernormal phase, a rare form of high grade partial A-V block is produced, that should be differentiated from complete A-V block

When an impulse that produces a P wave fails to elicit a ventricular response interference from an ectopic nodal or ventricular beat must be excluded before arriving at a diagnosis of A-V block The disturbed auriculoventricular conduction that occurs when a sinus beat reaches the junctional tissues immediately after an ectopic nodal or ventricular impulse is termed interference rather than A-V block Such interference is almost always recognizable from the presence of an ectopic QRS either immediately ahead of the blocked P wave, merged with it, or following it by an interval shorter than the customary P-R measurement Difficulty arises in the rare instance of the premature nodal impulse that fails to pass backward into the auricles and also fails to activate the ventricles, by reason of arrival before they have recovered from the preceding contraction Such a premature nodal impulse will itself cause no deflection, but will render the junctional tissues refractory to a sinoauricular

impulse that arrives immediately afterward resulting in an isolated blocked P wave in a rhythm that is otherwise regular * This rare phenomenon may be suspected when the tracing shows other premature nodal beats that give rise to retrograde P waves and/or premature QRS complexes of normal configuration If interference can be excluded as the reason for P waves failing to elicit a QRS response A-V block is established

The diagnosis of partial A-V block will be taken up first and the differentiation from complete block will be considered in connection with the description of the latter The electrocardiographic features of partial A-V block depend upon the degree of block This is expressed as a ratio between the number of auricular and ventricular complexes and may be classified into three grades

1) Low grade partial A-V block is manifested by intermittent *dropped ventricular* beats that are less frequent than the conducted beats *Thus, the most severe degree* of block arbitrarily classified into this category is that in which the average ratio of auricular to ventricular beats is 3 2 whereas successively diminishing degrees of block are expressed by ratios of 4 3, 5 4, 6 5, 7 6 8 7 or by notation of occasional dropped [illegible] ular beats may be ma[illegible] common and is usually [illegible] y go down from 4 3 to [illegible] † Whenever possible the role of vagal hyperirritability should be evaluated by repetition of the tracing after change from a recumbent to an erect posture, after exercise, or after a therapeutic dose of atropine Low grade partial A-V block may be subdivided into the following three groups according to the P-R relationships in successive cycles

   a) Essentially constant P-R interval apart from the dropped beats, is the uncommon finding and has been designated the Moebius type The P-R interval is prolonged but constant in all conducted cycles so that the dropped beat occurs abruptly The ventricular rhythm is regular except for intermittent pauses exactly twice the usual cycle length during which a regular sinus P wave is recorded but no QRS This form of A-V block should not be confused with S-A block in which no evidence of auricular activity is recorded during the pause or with a blocked premature auricular beat recognized by the prematurity and aberrant form of the isolated P wave

   b) [illegible] culminating in a dropped beat known as [illegible] ore common finding There is a progres- [illegible] R interval in successive cycles reflecting increasing fatigue of the junctional tissues until an auricular impulse fails to reach and activate the ventricles In the cycle following the dropped beat the P-R interval reaches its minimum and at that time is generally slightly above normal but may be within the normal range The increment in P-R is usually greatest in the second cycle following the dropped beat but the interval continues to lengthen by smaller increments until the next dropped beat which is prone to occur after the P-R has attained a length of 34 to .40 sec

   The pauses occasioned by the dropped ventricular beats are less than twice the usual cycle length and along with the irregularities in the intervening ventricular rhythm may produce a waning and waxing effect reminiscent of sinus

*When the sinoau[illegible]ular impulse arrives during the period of partial refractoriness induced by the blocked premature nodal bea[illegible] he sinoauricular P wave may elicit a ventricular response after a delay resulting in the prolongation of the P R interval in a single cycle a rare phenomenon to be distinguished from first degree block

†When a 2 1 ratio is maintained for more than a brief interval the block is classed as high grade

arrhythmia In the latter, the phasic changes in ventricular rhythm are dependent upon *similar changes in auricular rhythm* the P-R interval remaining essentially constant in the former, the phasic changes in ventricular rhythm result from abnormal and variable P-R intervals, the P-P intervals usually remaining *essentially constant*

The ratio of auricular to ventricular beats is seldom maintained at a fixed figure (such as 5 4) for more than a few minutes, but tends to vary in accordance *with fluctuations in vagal tone* *The role of the vagus should be investigated* either by attempts at stimulation, that would increase the degree of block, or at depression of vagal tone by change from recumbent to erect posture, by *exercise or therapeutic doses of atropine* *When the Wenckebach phenomenon is* of vagal origin, acceleration of auricular rate by exercise may lessen the degree of block or abolish dropped beats, when secondary to organic lesions of the A-V *node acceleration in auricular rate is prone to increase the degree of block*

c) Paradoxical shortening of a prolonged P-R interval preceding the dropped beat is a rare finding and may occur when the succeeding impulse fortuitously arrives at the A-V *node during its supernormal phase of recovery from the preceding* impulse This phenomenon should be suspected in partial A-V block when the P-R interval in the latter of two successive beats *is less prolonged* than in the *in the former provided that the auricular rhythm is regular and the QRS com*plexes uniform in contour The last stipulation is made to exclude premature ventricular beats but does not rule out an ectopic nodal beat released just *ahead of the arrival of the sinoauricular impulse* *To establish the diagnosis,* it is necessary to find paradoxical shortening of the P-R repeatedly through the tracing and to demonstrate a critical relation between the length of the P-R interval and that of the preceding R-P interval *Short R-P intervals falling* within a narrow range critical for the individual patient are followed by short P-R intervals presumably because of the arrival of the impulse during the *supernormal phase of recovery from the preceding* beat, whereas longer R-P intervals are followed by longer P-R intervals presumably because of arrival after the supernormal phase

2) High grade partial A-V block is manifested by dropped beats as numerous or more frequent than the conducted beats The common variety is 2 1 block in which every other P wave is followed by a QRS at a constant P-R interval The P-R interval of the conducted beat is usually *slightly prolonged* but *may fall within the* upper limit of normal The diagnosis is made by identification of the P wave with blocked ventricular response This P wave is detected by search *for deformity* in the *preceding S-T segment or T wave and is identified by its shape* and time relationships with other P waves as shown with the aid of dividers The recognition of the blocked P wave avoids mistaking 2 1 block for sinus bradycardia Further*more auricular ectopic beats so premature* that they fail to elicit a ventricular response, should not be confused with A-V block and are differentiated by their prematurity and significant differences in contour from P waves of sinus origin

In 2 1 block the P-P cycles may be perfectly regular or may alternate in length by as much as 08 sec to 12 sec In the latter event the cycles containing the QRS-T complex are *shorter* and the post-systolic cycles are lengthened The alternation in cycle length is dependent upon variations in vagal tone as shown by the fact that it is abolished by atropine The post-systolic slowing represents a reflex vagal retardation of impulse formation in the S-A node apparently induced by ejection of blood into the aorta Alternate P waves may differ in shape as well as in spacing presumably because of shift in the point of release from the S-A node

Fluctuations in vagal tone will also influence the ratio of auricular to ventricular beats in high grade partial block Reduction in *vagal tone may reduce the frequency*

of dropped beats resulting in a change from a 2 1 ratio to a lower grade 3 2 or 4 3 block or even to a 1 1 response whereas increase of vagal tone may convert a 2 1 block to a 3 1 or 4 1 block High grade partial block of the latter varieties is differentiated from complete A-V block by the demonstration of regularly recurring cycles in which a P wave is followed by a QRS at a constant P-R interval As long as the auriculoventricular ratio is constant in partial A-V block the auricular rate is a multiple of the ventricular

3) P cal

time for P waves to fall in order to elicit a ventricular response is between the summits of the T and U waves of the preceding beat representing an interval of 40 sec to 75 sec from the beginning of the last cycle in most reported cases P waves that fall before or after the critical period fail to elicit a ventricular response

When most P waves fall outside of the critical interval an independent (escaped) nodal or ventricular rhythm is necessarily set up and the tracing is likely to be mistaken for complete A-V block The differentiation is made by the demonstration that all P waves that fall within the critical period are followed at constant P-R intervals, by QRS complexes that are out of step with those of the independent rhythm This phenomenon may be explained in one of three ways and is not necessarily due to the same mechanism in every case

a) Supernormal phase of recovery According to this hypothesis A-V conduction is possible only when the impulse reaches the A-V node during the supernormal phase of recovery following the preceding impulse This explanation accounts for the observation in several cases that a 1 1 response occurred when the auricular rate was adjusted so that all P waves fell between the summits of the T and U waves of the preceding cycle but a complete block appeared when the auricular rate was slowed sufficiently to make all P waves fall after the U wave

b) Prolongation of rest period in the area of block According to this hypothesis antegrade conduction through the damaged A-V node takes place only after the preceding sinoauricular impulse has been extinguished proximal to the injured area thereby allowing additional time for recovery This takes place when an ectopic impulse is released from a focus in the ventricles or A-V node below the point of injury in time to permit retrograde conduction through the junctional tissues to meet and extinguish the downcoming sinus impulse above the area of injury This hypothesis has been supported by the demonstration of conduction from the ventricles back into the auricles to produce a retrograde P wave

c) Trans... ntric- A-V

c Complete block of forward conduction from auricles to ventricles (third degree block) is almost always but not invariably accompanied by interruption of retrograde conduction from ventricles to auricles this necessitates subdivision into two categories

1) Com... nduction ... the capac... for the ventricles to release impulses at an adequate rate for an indefinite period without interruption Serious heart disease is common but not invariable as evidenced by the reports of asymptomatic complete A V block of many years' duration Ventricular standstill with Stokes Adams

syndrome* is more common when the ectopic pacemaker is located in the ventricles than in the A-V node, and is also prone to occur at the advent of complete A-V block † The paroxysmal form may be diagnosed when serial tracings reveal episodes of complete A-V block and episodes of sinus rhythm with 1 1 A-V ratio The electrocardiographic features of complete A-V block are

a) Spacing and contour of the P waves When the auricles are under sinus control, the P-P intervals are usually regular however, variations in P-P intervals may occur due to the influence of reflexes, arising in connection with the independent ventricular activity, on the S-A node

In some cases, the auricular cycles enveloping a QRS-T complex are shorter than the cycles containing no QRS-T apparently because of preponderant influence of the aortic reflex initiated by ejection of blood Under these circumstances, the P wave following a QRS-T complex appears premature, but is recognized as of sinus rather than ectopic origin by a configuration identical with other P waves

When there are three or more successive P waves without an intervening ventricular contraction the P-P intervals may exhibit progressive shortening owing to the Bainbridge reflex, arising from distended atria During prolonged ventricular standstill the auricular rate first accelerates then progressively slows If ventricular standstill lasts more than 20 sec the P waves are likely to become bizarre and polymorphous

b) Ventricular rate is usually between 30 and 40 beats per minute It may exceed 40 particularly when digitalis intoxication is an etiologic factor or may be less than 30 and even as low as 10 beats per minute The ventricular rate is always slower then the auricular This is the primary criterion in differentiating complete A-V block from interference dissociation, in which the ventricular rate is faster than the auricular The ventricular rate is often approximately one-half of the auricular but accurate measurement of the respective rates shows lack of arithmetical relationship This aids in the differentiation from constant 2 1 A-V block in which the ventricular rate is exactly one-half of the auricular

c) Ventricular rhythm

(1) Regularity depends upon whether there is a single or multiple pacemaker for the ventricles When there is a single pacemaker as indicated by uniformity in QRS contour in a given lead the ventricular rhythm will be regular unless there are episodes of standstill This is in contrast to low grade partial A-V block in which the ventricular rhythm is always irregular When there is more than one pacemaker for the ventricles in complete A-V block the rhythm is irregular In this event QRS complexes in a given lead differ in contour as well as in spacing but consecutive beats from the same focus are evenly spaced and uniform in pattern

(2) Independence from the auricular rhythm is established by demonstrating (1) that the respective rates are arithmetically unrelated (2) that the ventricles fail to respond to any sinoauricular impulses, regardless of the portion of the cycle in which the P wave falls This constitutes the fundamental criterion for the differentiation of complete A-V block from both partial A-V block

*The Stokes Adams syndrome characterized by syncopal attacks during which the auricles continue to beat but the ventricles fail to maintain circulation may result not only from ventricular standstill secondary to complete A V block but also from paroxysmal ventricular tachycardia

†Intermittent complete A-V block is usually referable to transient ischemia complicating coronary disease occasionally to strong vagal stimulation rarely to other causes such as recurrent inflammation

and interference dissociation When antegrade conduction is completely interrupted a study of a long continuous record of one lead or many shorter strips from multiple leads will show P waves at all possible points between the QRS complexes In the presence of complete A-V block no P wave regardless of its time in respect to the ventricular cycle, will elicit a ventricular response and thereby break the regularity of the independent ventricular rhythm

d) Location of the ventricular pacemaker The electrocardiographic findings and the clinical course depend, to a considerable extent upon whether the pacemaker is situated in the lower part of the A-V node or in one of the ventricles With a nodal pacemaker the resting ventricular rate is usually in the range of 35 to 45 beats per
of standstill With
erally slower tra
and the prognosis i
judged from the QRS pattern

(1) P

... block may be indistinguishable from an idioventricular pacemaker unless the bundle branch block is intermittent This is recognized by shifting between QRS patterns of normal and prolonged duration in the presence of a perfectly regular rhythm A nodal pacemaker with bilateral bundle branch block has been diagnosed by the demonstration of even spacing of the QRS complexes with alternation between patterns of left and right bundle branch block type

(2) Idioventricular pacemaker is necessitated by lesions involving the A-V node and both branches of the bundle of His The QRS-T complex is broad and bizarre and is subject to changes in configuration as a result of the tendency for the idioventricular focus to shift in location The approximate site of the pacemaker is judged by the criteria employed for locating the focus for ectopic ventricular beats (page 106)

2) Complete forward block with preservation of retrograde conduction a rare phenomenon To make the diagnosis the presence of complete forward block must be established first The preservation of retrograde conduction is indicated by the demonstration of intermittent P waves of opposite direction to those of sinus origin (i e upright in aV_R inverted in aV_F) following ...

... Retrograde passage at other ... cycle is blocked by the downcoming sinus impulse The interval between the QRS and the succeeding retrograde P wave generally ranges between 10 and 23 sec

2 Accelerated conduction from auricles to ventricles is manifested by a P-R interval less than 12 sec in duration Before the diagnosis is made, it is necessary to exclude nodal rhythm described on page 110 Sinus mechanisms with short P-R intervals may be classified into two types depending upon the pathway of conduction from auricles to ventricles along with the manner of ventricular excitation

a Accelerated conduction through the customary pathways with normal ventricular excitation is manifested by P waves consistent with an S-A pacemaker a P-R interval

slightly less than 12 sec , and QRS complexes of normal configuration and duration This combination represents the usual finding at birth, it is not uncommon in children under thirteen years of age presumably because of the small size of the heart, and is occasionally observed in adults The P waves tend to have a church steeple contour with narrow base suggesting rapid auricular activation, but originate from a pacemaker in the S-A node, or at least in the immediate vicinity * as shown by complete inversion in Lead aV_R and an upright deflection in aV_F This syndrome has been observed not only in normal adults, but also in hypertensive cardiovascular disease and in adrenal cortical hyperfunction A predilection towards paroxysmal tachycardia has been reported

b Accelerated conduction through aberrant pathways with anomalous ventricular excitation (Wolff-Parkinson-White syndrome) The bundle of Kent is a congenital anomaly in humans with predilection for (1) the right lateral aspect of the heart, bridging the groove between right atrium and ventricle, or (2) the posterior wall connecting either the left atrium and ventricle the right atrium and ventricle, or the posterior ends of the septa The bundle may never function, it may conduct impulses in episodes alternating with periods during which the ventricles are under the exclusive control of impulses transmitted through the A-V node In the presence of a bundle of Kent, episodes of W-P-W syndrome are prone to occur even though the heart is otherwise normal or may be precipitated by lesions that cause greater impairment of conduction through the A-V node (e g rheumatic and diphtheritic myocarditis septal infarction) etc

The probable genesis of the W-P-W syndrome in a patient with a functioning right lateral bundle is illustrated in Fig 23 A B The shortening of the P-R interval by the premature onset of the QRS results from transmission of the auricular impulse through the bundle of Kent to the right ventricular surface prior to its passage through the atrioventricular node (Fig 23 A) The premature component is downward in direction in leads from the right precordium and axilla (e g V_{3R}) which reflect the potential variations of the epicardial surface of the lateral wall of the right ventricle, away from which the impulse is moving, it is upright in leads from the left side of the chest (e g V_3 V_6) towards which the impulse is advancing as indicated by the arrows in Fig 23 A The relatively slow progress of the impulse along aberrant pathways is responsible for the thickening and slurring of the premature component and for the lengthening of the QRS duration The abrupt transition from a deformed initial phase to a normal final phase of the QRS presumably results from the extinction of the aberrant impulse in the right ventricle by the arrival of the normal impulse distributed through the ramifications of the bundle of His with activation of the remainder of the ventricles in the usual manner (Fig 23 B) Since ventricular depolarization is completed in the normal manner the time interval from the beginning of the P to the end of the QRS in patients with intermittent W-P-W syndrome is generally the same in the anomalous as in the normal cycles The electrocardiographic features of the Wolff-Parkinson-White syndrome are as follows

1) P waves are characteristically normal in direction contour and duration and simulate those of normal sinus mechanism in cases of intermittent W-P-W syndrome Anomalous atrioventricular conduction can complicate ectopic auricular rhythms and has been reported in association with auricular premature beats auricular fibrillation auricular flutter and nodal rhythm Although the presence of the W-P-W syndrome may be suspected from the nature of the QRS deformity a

*This syndrome has been designated as coronary sinus rhythm but it seems unlikely that the pacemaker is located in this vicinity or in the upper part of the A V node in view of the direction of the P waves in the unipolar limb leads

positive differentiation from aberrant conduction through the ramifications of the bundle of His generally awaits the identification of the W-P-W complexes after reversion to normal sinus rhythm

2) P-R interval is shortened as a result of obliteration of the P-R segment by the premature onset of the QRS and is characteristically less than 12 sec Measurements may vary in different leads The shorter values are obtained in leads nearest the portion of the ventricles initially activated by way of the bundle of Kent and

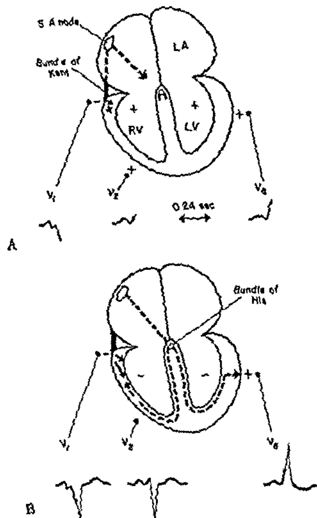


Figure 23

should be taken as the true measurement provided there is no reason to suspect that the first portion of the P wave is isoelectric Erroneously long measurements amounting to 12 sec or more may be obtained in distant leads so far removed that they fail to register the potential differences developed at the beginning of anomalous ventricular excitation

P R intervals exceeding 12 sec in all leads may be found in the event of (1) prolongation in the duration of the P wave to 13 sec or more as the result of an intraauricular conduction defect (2) retardation in passage of the impulse through the bundle of Kent but still greater delay in transmission through the atrioventricular node The short P R interval of the W P-W syndrome must be differentiated from the short P-R interval associated with A-V nodal rhythm and that associated with accelerated conduction through the normal pathways (described

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b / t i

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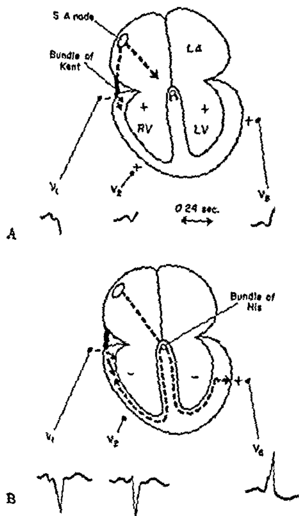


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on page 99) In W-P-W syndrome the P waves are characteristically of sinoauricular origin (i e , upright in Lead aV_F, inverted in aV_R), and the QRS shows the classical deformity of its initial phase, in auriculoventricular nodal rhythm, the P waves are opposite in direction (i e , inverted in Lead aV_F, upright in Lead aV_R), and are generally followed by QRS complexes of normal contour, in accelerated conduction through the normal pathways, the P waves have a characteristic narrow base and steeplelike contour and the QRS complexes are generally normal in contour

3) QRS duration usually is prolonged beyond 10 sec and ranges between 11 and 18 sec The prolongation occurs in the portion of the QRS inscribed prior to the onset of the intrinsicoid deflection This interval exceeds 05 sec and generally ranges between 06 and 12 sec depending upon the length of the premature component The greater the duration of the QRS and the premature component the greater the distortion of the QRS-T contour, due to the registration of relatively more premature and relatively less final component The lengthening of the QRS occurs at the expense of the P-R interval Measurements of the time between the onset of the P and the end of the QRS (P-J interval) are generally the same in anomalous as in normal complexes when both are present in the same tracing, however the P-J interval may be shorter in anomalous complexes when the difference between the time of arrival of the impulse at the ventricular terminus of the bundle of Kent and A-V node is great enough to permit complete activation of both ventricles through the aberrant pathway This is prone to occur when the impulse distributed through the bundle of Kent reaches the ventricle 06 sec or more ahead of that passing through the A-V node to the bundle of His

4) QRS contour The classical QRS in the W-P-W syndrome is a fusion complex, consisting of (1) a premature component (delta wave) characterized by a relatively thick string image with either slurring or notching reflecting a comparatively slow progress of ventricular activation through an anomalous pathway, (2) a final component characterized by a thin string image and a precipitous deflection representing a comparatively rapid movement of the activating impulse distributed through the bundle of His and its ramifications The relative duration and magnitude of the premature and final components are dependent upon the duration of the interval between the arrival of the impulse transmitted through the anomalous pathway and that conducted through the A-V node and bundle of His, as discussed above The premature component is brief in duration and small in amplitude when the impulse spreading from the ventricular terminus of the anomalous pathway is soon extinguished by the arrival of that distributed through the bundle of His it is longer in duration and greater in amplitude when there is greater disparity in onset of ventricular activation through the two pathways The premature component forms the entire QRS when the impulse transmitted through the anomalous pathway arrives sufficiently early to activate all the ventricular myocardium Under these circumstances the QRS is not a fusion complex but rather resembles an ectopic ventricular beat arising from a focus corresponding with the ventricular terminus of the anomalous pathway

5) QRS direction The direction of the premature component of the QRS in a given chest lead depends upon the location of the anomalous pathway in reference to the heart and to the position of the electrode When the bundle of Kent bridges the gap between the lateral aspect of the right atrium and ventricle the impulse arrives in the subepicardial aspect of the lateral wall of the right ventricle and proceeds to activate the ventricle in the right-to-left direction Under these circumstances the premature component will be recorded as a downstroke in leads from the right precordium and axilla which face the epicardial surface of the lateral wall of the right ventricle (V_1 to V_{5R} inclusive) and as an upstroke in leads from the left side of

the chest (V_2 to V_9 inclusive) When the anomalous pathway extends between the posterior aspect of left atrium and ventricle the impulse arrives in the postero-basal surface of the left ventricle to activate the ventricles in a dorsoventral direction (Fig 24) Under these circumstances an initial downstroke will be recorded

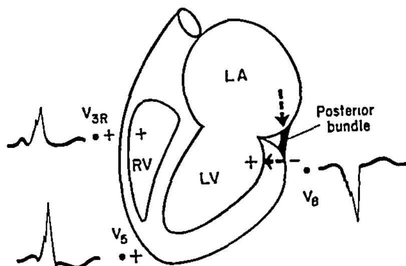


Figure 24

in leads from the posterior wall of the chest (V_8 V_9 V_{9R} and V_{8R}) and an upstroke in leads from the anterior aspect of the right side of the chest (V_1 to V_{5R} inclusive) as well as from the left precordium (V_2 to V_6 inclusive)

The differentiation between a right lateral bundle and a posterior bundle as the anomalous pathway is made from the direction of the premature component in leads from the right precordium (V_{5R} V_{4R} V_{3R} and V_1) as compared to that in leads from the back (V_8 V_9 and V_{9R}) The registration of an initial downstroke in Leads V_{6R} V_{4R} V_{3R} and V_1 and an initial upstroke in Leads V_8 V_9 and V_{9R} of the same case indicates that the vector associated with anomalous excitation is directed from right to left in a transverse plane and from front to back in a sagittal plane and thus points to activation through a bundle of Kent terminating in the subepicardial aspect of the lateral wall of the right ventricle The registration of an initial downstroke in Leads V_8 V_9 and V_{9R} and an initial upstroke in Leads V_{6R} V_{4R} V_{3R} and V_1 of the same case indicates that the vector associated with anomalus excitation is directed from left to right in a transverse plane and from back to front in a sagittal plane and thus points to activation through a bundle of Kent terminating in the subepicardial aspect of the posterobasal wall of the left ventricle

Esophageal leads opposite the atrioventricular groove like Leads V_8 and V_9 show a premature upstroke when the anomalous pathway is on the right lateral aspect of the heart and a premature downstroke when the anomalous pathway is posterior On the other hand esophageal leads opposite the posterior diaphragmatic aspect of the left apex (E_{55} and E_{60}) as well as precordial leads facing the anterolateral aspect of the apex (V_4 V_5 and V_6) display a similar premature upstroke when anomalous excitation is through a lateral or posterior bundle of Kent presumably because the direction of the vector in a cephalocaudad plane is from base towards apex in either case

6) S T complex and T wave The findings are quite variable and depend in part on the relative proportions of ventricular activation by way of the anomalous pathway and the Purkinje system When the premature component is brief the S-T junction tends to be isoelectric and the T wave normal in direction The more

prolonged and larger the premature component the more likely the displacement of the S-T segment and T wave in a direction opposite to the QRS The T wave is also subject to considerable variation in leads facing different portions of the same ventricle in a given patient No inference regarding the state of the myocardium can be drawn from the QRS-T pattern when the W-P-W syndrome is present

7) Paroxysmal tachycardia is a common complication and was present in approximately two-thirds of the cases in the largest individual and collected series The true incidence is no doubt, below this figure since asymptomatic patients tend to remain undiscovered During the paroxysm the P waves resemble those of auricular tachycardia and along with the associated QRS complexes occur at a rapid rate believed referable to the establishment of a continuous circuit between auricle and ventricle The QRS complexes are generally of normal supraventricular form attributable to antegrade conduction of the circular impulse through the auriculoventricular node and retrograde conduction through the bundle of Kent Upon cessation of the tachycardia, the QRS complexes revert to the fusion pattern typical of the W-P-W syndrome On the other hand the QRS complexes during the paroxysm may resemble those of ventricular tachycardia presumably from a reversed circuit Under these circumstances a supraventricular origin is indicated by a constant and uniform relation to the P waves An abnormally short P-R interval would suggest a reversal in circuit with conduction from atrium to ventricle through the bundle of Kent a normal P-R interval would suggest transmission through the auriculoventricular node with complicating bundle branch block to explain the bizarre ventricular complexes Paroxysmal auricular fibrillation or flutter is responsible for the episodes of rapid heart action in a few cases Some of the ventricular complexes may be normal and others fusion in type indicating conduction through both pathways

8) Influence of physiological factors and pharmacological agents The W-P-W syndrome is often intermittent and is sometimes manifested by alternation between normal and fusion QRS complexes Reversion to sinus rhythm may occur during exercise presumably as a result of vagal inhibition with consequent acceleration of transmission through the auriculoventricular node Digitalis and cholinergic drugs (neostigmine and methacholine) given at a time when conduction is normal cause reappearance of the W-P-W syndrome and exaggerate the effect of a pre-existing anomalous excitation, presumably through lengthening conduction time through the A V node and allowing more of the ventricles to be activated by the impulses transmitted through the anomalous pathway Digitalis may fail to control ventricular rate in patients with auricular fibrillation when the ventricles are activated through the bundle of Kent Atropine has abolished the W-P-W syndrome in a few cases but more often has been of no value Quinidine is the most effective therapeutic agent and presumably acts by delaying conduction through the bundle of Kent sufficiently to permit ventricular excitation through the ramifications of the bundle of His Paroxysmal tachycardia associated with the W-P-W syndrome may be converted to sinus rhythm by quinidine

D SINUS RHYTHM (OR ARRHYTHMIA) COMPLICATED BY ECTOPIC BEATS The classification according to site of the ectopic pacemaker (i e whether nodal auricular or ventricular) and according to spacing (i e whether a premature or an escaped beat) has been discussed at the beginning along with the pathogenesis, thus leaving merely a description of the electrocardiographic features

1 Premature ectopic beats are recognized by the interjection into a sinus rhythm of beats that are (1) premature, as shown by their occurrence in advance of the next anticipated cycle, (2) ectopic as shown by significant differences from P and/or QRS deflections

in cycles of sinus origin * Premature beats may be occasional or frequent may occur singly in pairs or short runs They may occur after each sinus cycle causing a bigeminal (coupled) rhythm or after each pair of sinus cycles resulting in a trigeminal rhythm They may arise from a single focus as indicated by a consistent shape in any given lead or from more than one focus as recognized from decided differences in contour between two or more premature beats in the same lead Premature beats from a single focus show a constant time relation to the preceding sinus cycle as would be expected if they were an expression of re-entry discussed on page 85 They may be classified according to location of the focus as supraventricular (nodal or auricular) or ventriuclar and each group may be further subdivided in accordance with contour and/or spacing of the premature P and/or QRS complexes

a Premature supraventricular beats are recognized from the interjection of premature ectopic P waves into a basic rhythm originating from the sinoauricular node Premature supraventricular beats may arise from any part of the atria or A-V node The approximate source is determined from the contour and spacing of the premature P waves the manner of conduction through the A-V node and ventricles as indicated by the P R interval and the QRS-T contour as compared with sinoauricular cycles

1) Contour of the premature P waves Evaluation of contour or even recognition is often handicapped by the tendency for premature P waves to be superimposed upon the T waves of the preceding cycle The P wave often appears as a notch that is easily identified and evaluated however the P wave may be perfectly fused with the T wave in which event it is recognized and evaluated as to direction and amplitude by comparison of the fused complex with other pure T waves The location of the focus is judged from the direction and shape of the premature P waves particularly in the unipolar limb leads as amplified below The presence of two or more ectopic foci is indicated by significant differences in the shape and/or direction of the ectopic P waves in a given lead The conductivity of the impulse through the remainder of the atria is determined from the duration of P wave as discussed on page 31

a) Nodal premature beats are manifested by ectopic P waves that are sharply inverted and V shaped in aV_F and upright in aV_R and aV_L

b) Auricular premature beats When the focus is located in the right atrium the P wave is inverted in Lead aV_R but differs significantly in contour and amplitude from the P wave of sinus origin it is usually upright in aV_L and aV_F but may be shallowly inverted in the former when the focus is in the upper part of the right atrium or shallowly inverted in the latter when the focus is in the lower portion P waves arising from the left atrium are upright in aV_R if the focus is high in the atrium they are inverted in aV_L and upright in aV_F if the ectopic site is low they may be reversed in direction in the two latter leads

2) Spacing of the premature P waves

a) P P intervals The interval between the preceding sinus P wave and the ectopic P wave is shortened whereas the interval between the ectopic P wave and the next sinus beat is lengthened After nodal beats this pause usually compensates for the prematurity of the preceding cycle after auricular beats the pause is usually insufficient for full compensation

Premature auricular beats are characterized by premature P waves that show significant differences in contour from the customary P waves They are followed by QRS T complexes of either customary or aberrant form Premature ventricular beats are characterized by premature QRS T complexes of bizarre contour The P wave is almost always unaltered but may be replaced by a retrograde P wave when the ventricular beat is very premature

b) P-R intervals

(1) Nodal premature beats The P-R interval depends upon the portion of the node in which the focus is located and the relative speeds of retrograde and forward conduction If the focus is in the upper portion of the node the aberrant P may precede the QRS by an interval of 02 to 12 sec , if lower down the aberrant P wave may be submerged in the QRS or may follow it In the event of retrograde block a P wave of sinus origin may be detectable just before, in or just after the QRS the nodal origin for the latter being indicated by a contour like that of the QRS-T complexes occuring in response to a sinoauricular impulse, but premature in time

(2) Auricular premature beats The P-R interval may be similar to that of the associated sinus beats, or slightly longer or shorter depending in part on the location of the ectopic focus on the degree of prematurity and on the conductivity of the A-V node When very premature, the P-R interval may be significantly prolonged or a QRS response may fail to occur, due to partial or complete refractoriness of the junctional tissue In the latter event an isolated premature P wave is found

(3) Ventricular response A premature P wave should be identified to establish the fact that the impulse responsible for the QRS-T arises from above rather than from within the ventricles QRS-T complexes of premature supraventricular impulses may be identical with those of sinus origin or may differ very slightly in height shape or duration When the P wave is quite premature the impulse may reach the ventricles before they have fully recovered from the previous contraction Under these circumstances the impulse may be blocked or may take an aberrant course through the ventricles resulting in a bizarre QRS-T complex resembling that of premature ventricular beats When the ectopic P wave is very premature the bizarre QRS-T response may be found, irrespective of whether the ventricles are normal or abnormal when it is only slightly premature, a bizarre QRS-T response raises the question of a latent conduction defect within the ventricles

b Premature ventricular beats are recognized from the interjection of premature bizarre QRS-T complexes into a basic sinus (or ectopic) rhythm along with the exclusion of premature ectopic P waves The QRS complexes of premature ventricular beats are invariably longer in duration and different in shape from those of supraventricular origin They usually exceed 11 sec in duration and are coarsely slurred or notched The T wave is opposite in direction to the main deflection of the QRS Premature ventricular beats may arise from any portion of either ventricle The approximate focus is determined from the comparative contour of the QRS in multiple chest leads Premature ventricular beats are further characterized by their effect on auricular and ventricular rhythm

1) Contour serves as an index of the number of foci and the approximate location of each focus when premature beats are frequent A single focus is manifested by uniformity in contour of all premature beats in each individual lead more than one focus is present when two or more premature beats in the same lead show decided differences in direction and/or contour The approximate location of the focus is determined from the QRS patterns in multiple chest leads A QS complex is recorded in the lead or leads overlying the epicardial surface of the ventricular segment from which the premature beat originates a monophasic R wave is obtained in leads over the opposite wall of the heart a diphasic RS complex is registered in intervening leads The upright component of this RS complex is small and brief in duration in leads over portions of the ventricle near the ectopic focus

and increases in amplitude and duration at the expense of the S wave in leads farther and farther removed from the site of origin

2) Spacing

a) Relation to preceding ventricular cycle All ventricular premature beats that come from the same focus and occur singly have a constant temporal relation to the antecedent ventricular cycle The time of predilection for ventricular premature beats is early in diastole the ectopic QRS being superimposed on the U wave of the preceding cycle (i e the phase of supernormal excitability) The other common type of ventricular premature beat occurs late, superimposed on the P wave or following it by an interval significantly shorter than the customary P-R Under the latter circumstances the QRS complex may have the features of a fusion beat (i e initial phase slurred final phase like that of supraventricular origin), due to activation of a portion of the ventricles through the ectopic focus the remainder through ramifications of the bundle of His

b) Auricular rhythm is usually undisturbed a sinoauricular P wave occurring at the scheduled time When the ventricular beat is only slightly premature the P wave may be superimposed upon the initial phase of the QRS or may precede it by an interval shorter than the customary P-R When the ectopic ventricular beat coincides with the U wave of the preceding cycle, the P wave will fall somewhere on the bizarre QRS-T complex and can be located by dividers Even though this P wave is completely buried in the ventricular complex its presence may be inferred by the demonstration, with the aid of dividers of an undisturbed auricular rhythm When the ventricular beat is very premature and the heart rate slow a retrograde impulse may pass from the ventricles through the A-V node and excite the auricles before the sinus node is discharged producing a retrograde P wave (inverted in aV_F upright in aV_R) in advance of the scheduled time for the sinus P wave The impulse under formation in the sinus node is extinguished and the sinus rhythm is interrupted for one beat When a retrograde impulse from the ventricles is on its way through the auricles at the time of arrival of an antegrade impulse from the sinus node, a fusion P wave reflecting a mixture of each is recorded beginning just ahead of the customary P wave

c) Ventricular rhythm Provided that the auricles are beating regularly under sinus control the interval between the ectopic and next supraventricular QRS is lengthened sufficiently to compensate exactly for the shortening of the preceding cycle The pause is noncompensatory under the following circumstances

(1) Retrograde auricular systole

(2) Interpolation of the ectopic ventricular beat between two normally spaced QRS complexes This occurs when a premature ventricular beat is blocked from further retrograde conduction at the A-V node and when the heart is so slow that the A V node and the ventricle recover in time to respond to the next impulse coming down from the auricle The cycle which follows an interpolated extrasystole is likely to show a prolonged P-R interval and a distorted QRS T This is the only type of premature ventricular beat which is a true extrasystole

(3) Underlying ectopic auricular rhythm (page 113)

3) Succeeding cycle The pause following a ventricular premature beat may be exactly compensatory or may be unusually long in which event it may be terminated by a sinus beat or an ectopic escaped beat When a compensatory or prolonged pause is terminated by a sinus beat the P wave and P-R interval are

identical with others in the tracing The resultant QRS-T is usually similar to others of supraventricular origin, but may differ, particularly in respect to the ST-T complex The T component of a cycle following a ventricular premature beat may differ significantly in amplitude and direction from the T waves in other supraventricular cycles not preceded by a ventricular premature beat When differences are present the more common finding consists of upright T waves in the customary sinus cycles and inversion in the sinus cycles following premature ventricular beats

2 Escaped ectopic beats Escape is recognized by (1) a preceding pause distinctly longer than the customary T-P interval, (2) termination of the pause by a beat identified as ectopic Escape constitutes a protection against undue cardiac standstill The pause that initiates escape may occur as a part of marked sinus arrhythmia, as a result of sinoauricular or partial A-V block or following a ventricular premature beat When a pause longer than the customary T-P interval is terminated by a single ectopic beat followed by resumption of normal sinus rhythm the phenomenon comprises isolates escape, when the undue pause is followed by a succession of ectopic beats from the same focus usually at a rate below 100 the phenomenon consists in an escaped ectopic rhythm

Escaped beats are recognized as supraventricular in origin when the pause is terminated by a QRS-T complex that is identical with or only slightly different from the QRS-T complexes resulting from a sinoauricular impulse, and as ventricular in origin when the pause is terminated by a broad, bizarre QRS quite different from that occurring in response to sinus impulses When the pause is terminated by a supraventricular escape the focus is usually in the A-V node, rarely in an atrium The differentiation and approximate localization of the focus is the same as for premature supraventricular beats given on page 105

Escaped nodal beats are further characterized by the type of auricular response (1) retrograde conduction is indicated by a P wave that is upright in aV_R inverted in aV_F and immediately precedes coincides with or follows the QRS (2) retrograde block may be presumed when no evidence of auricular activity is detectable, (3) interference is indicated when a P wave similar to that of sinus beats, is found just ahead of the QRS (at a P-R interval less than 12 sec) superimposed upon the QRS or following it

II ECTOPIC NODAL FOCUS may coexist with the sinus pacemaker, in which event it excites the ventricles and the sinus node governs the auricles or it may usurp control of the entire heart by retrograde conduction through the atria and antegrade activation of the ventricles

A COEXISTENT SINUS PACEMAKER AND ECTOPIC NODAL FOCUS OR PACEMAKER

The sinus and nodal pacemakers may be concurrent or consecutive as determined from a study of the entire tracing When concurrent each cycle shows evidence of P waves arising from sinoauricular impulses and QRS-T complexes originating from A-V nodal impulses When consecutive there is a series of beats in which the whole heart is controlled by impulses of sinoauricular origin interspersed with episodes during which the ventricles are taken over by the A-V node and the auricles either remain under sinus control or are activated by retrograde impulses from the A-V node

1 Concur ... falling into this category ... discussed above under another classification and certain arrhythmias that belong primarily under this heading

a Sinus rhythm with nodal beats and interference includes premature nodal beats discussed on page 105 and escaped nodal beats discussed above In cycles exhibiting interference the QRS-T complex is of nodal origin and is dissociated from the P wave of sinus origin

b Escaped nodal rhythm with retrograde block and interference dissociation When the rate of impulse release from the sinus node is depressed below the A-V node interference dissociation may occur The ventricles under A-V nodal control beat at a slow to normal rate but more rapidly than the auricles or they may beat at approximately the same rate but slightly in advance of the arrival of the sinoauricular impulse When the sinus impulse causing the P wave periodically arrives late enough that the junctional tissue and ventricle have regained responsiveness there is a periodic conducted beat interspersed between short episodes of escaped nodal rhythm a phenomenon known as intermittent interference dissociation When the sinus impulse causing the P wave always arrives just after the ventricles have responded to an impulse originating in the A-V node no impulses are conducted from the sinus node to ventricles a condition referred to as complete interference dissociation

1) Intermittent interference dissociation complicating an escaped A-V nodal rhythm with retrograde block The background for this unusual arrhythmia is (a) the presence of a pacemaker in the A-V node that generates impulses slightly faster than the depressed sinus node (b) the preservation of forward conduction but block of retrograde conduction through the A-V node The sinus node has exclusive control over the auricles as indicated by uniform P waves of sinus origin but competes with the A-V nodal pacemaker for control of the ventricles Periodically a sinoauricular impulse is conducted through the A-V node because of arrival slightly after the junctional tissue has recovered from the refractory state induced by generation of the preceding impulse The regularly spaced P wave produced by this impulse leads to an isolated ventricular response following a P-R interval of 12 sec or more

Upon re-establishment of ventricular control by the more active lower pacemaker the first QRS occurs abnormally early in respect to the P wave generally at an interval short enough (i e less than 12 sec) to indicate its independence from the P wave Subsequent QRS complexes occur at regular intervals shorter than the P P intervals As a consequence the P wave in succeeding cycles falls farther and farther backward in respect to the QRS (the so called reversed Wenckebach phenomenon) and finally occurs late enough that the sinoauricular impulse again reaches a responsive A V node through which it is conducted to resume ventricular control The QRS so produced appears early in respect to the preceding ventricular complexes (originating from the lower pacemaker) and may be mistaken for an auricular premature beat at first glance but is easily differentiated by the uniform P wave contour and spacing and the characteristic variations in P and QRS relationships during the period when the ventricles are under the domination of the lower pacemaker

2) Complete interference dissociation complicating an escaped nodal rhythm with retrograde block This rare arrhythmia requires (a) the presence of pacemaker in the A V node that generates impulses at approximately the same rate as the sinus node and sends them into the ventricles slightly in advance of the arrival of the sinoauricular impulse at the junctional tissue (b) the presence of a retrograde block As a consequence of these rigid requirements this disorder is always temporary but may last throughout the recording of an electrocardiogram

As long as the necessary relationships persist the disorder is manifested by (a) P waves of sinus type (inverted in aV_R upright in aV_F) that occur either immediately ahead of the QRS (with P R interval of less than 12 sec) superimposed upon the QRS or immediately behind the QRS (b) QRS complexes that are regularly spaced and usually normal in contour (nodal pacemaker) rarely bizarre in contour (ventricular pacemaker or nodal pacemaker with abnormal intraventricular conduction) The relationship of P to QRS tends to vary slightly in different parts of the tracing due to the fact that rates of the two pacemakers are seldom identical

When sufficient disparity in rates of the two pacemakers develops so that the P wave falls on or after the T wave, the sinoauricular impulse should be conducted to the ventricle to interrupt the interference dissociation and convert it to the intermittent variety discussed above

c Complete A-V block with ventricular activation from an A-V nodal pacemaker (and auricular activation from a sinus pacemaker), discussed on page 97

2 Consecutive sinus and A-V nodal pacemakers A study of the tracing reveals consecutive cycles during which the entire heart is activated by sinoauricular impulses interspersed with episodes during which the ventricles are activated by an A-V nodal pacemaker During these latter episodes the auricles may remain under sinus control resulting in intermittent interference dissociation discussed above (page 109) or the auricles may be activated in retrograde fashion from the A-V node This constitutes the condition known *as wandering pacemaker between the sinus and A-V nodes*

There is a single pacemaker for the auricles and ventricles, which shifts back and forth between the S-A and A-V nodes, as a result of vagal depression of the more irritable focus P waves of sinus origin (inverted in aV_R upright in aV_F) are replaced during the course of a few cycles by oppositely directed retrograde P waves and abnormally short P-R intervals of nodal origin During the transition fusion P waves intermediate between those of sinus and nodal origins are prone to occur as the result of simultaneous auricular activation by impulses from the S-A and A-V nodes When the pacemaker wanders back to the sinus node, the reverse changes in P waves and P-R intervals occur

B NODAL RHYTHM with replacement of sinus pacemaker The entire heart is activated by impulses released from the A-V node and conducted antegrade through the ventricles retrograde through the auricles On the basis of heart rate and mechanism, A-V nodal rhythms may be classified as (1) passive an escape phenomenon occurring when the sinus node is depressed more than the A-V node and manifested by a slow nodal rhythm below 100 per minute, (2) active or nodal tachycardia, arising from a hyperirritable focus with rates exceeding 100 and usually above 150

1 Passive A-V nodal rhythm with retrograde auricular conduction is manifested by evenly spaced uniform P waves that are upright in Leads aV_R and aV_L and inverted and V-shaped in aV_F occurring at a rate below 100 The QRS complexes are normal unless there is a complicating ventricular lesion Further subdivision is made in accordance with the temporal relationships of the retrograde P and the antegrade QRS

a Upper nodal rhythm – P wave precedes QRS, usually by an interval of less than 12 sec but beyond 12 sec in the presence of delayed A-V conduction

b Middle nodal rhythm – P wave is superimposed upon QRS

c Lower nodal rhythm – P wave follows QRS after a variable interval depending upon the site of the pacemaker and the speed of retrograde conduction

1) Reciprocal rhythm a rare phenomenon occurring in lower nodal rhythm and recognized by a second QRS following the retrograde P simulating the first QRS in contour and separated from it by an interval of 5 sec or less The production of a second ventricular (reciprocal) response has been attributed to the establishment of a circuit through the A-V node whereby the impulse moving backward through a portion of the A-V node to stimulate the auricles reverses and moves forward through another portion of the A-V node to reach and activate the ventricles for a second time Rarely the circuit through the A-V node may continue and thus produce a third and even a fourth ventricular response

2 Nodal tachycardia – to be discussed along with auricular tachycardia as a form of supraventricular tachycardia (page 111)

III ECTOPIC AURICULAR FOCUS may coexist with the sinus pacemaker and intermittently control the atria and ventricles or may replace the sinus pacemaker, usurping control over the *entire heart*

A COEXISTENT SINUS PACEMAKER AND ECTOPIC AURICULAR FOCUS OR PACEMAKER This category includes sinus *rhythm* with ectopic auricular beats more appropriately classified above under another heading (page 105) and a very rare disorder auricular parasystole

1 Auricular parasystole is manifested by two sets of P waves, different in configuration slow in rate and independent in rhythm One set is of sinus origin and the other arises from an ectopic focus isolated from effects of the sinus impulse by a *protective block* The latter set of P waves is likely to be mistaken for auricular premature beats at first glance but is distinguished by the following characteristics (1) a varying temporal relationship to the preceding cycle of sinus origin as contrasted to the constant relationship characteristic of repeated auricular premature beats from a single focus, (2) a constant but slower rhythm than that of sinus origin, which is best demonstrated with the aid of dividers Gaps of an exact multiple of the parasystolic interval may occur when the ectopic focus happens to discharge at a time when the auricles are responding to the sinus impulse Likewise gaps in the sinus rhythm may occur when the sinus node discharges during or shortly after an auricular response to the parasystolic focus When each set of P waves is coarsely notched and 12 sec or more in duration an organic atrial lesion causing *intra-atrial block* may be responsible for the parasystolic rhythm

B ECTOPIC AURICULAR RHYTHM replacing sinus pacemaker When the sinus node is depressed there is an escape to a lower pacemaker usually the A-V node occasionally an ectopic focus in an auricle or ventricle An escaped auricular rhythm is manifested by a slow regular rhythm (below 100) originating from an ectopic focus in an auricle not the A-V node as judged from the contour of the P wave On the other hand the active ectopic auricular rhythms which are much more common are an expression of hyperirritability and usurp the pacemaker The pathogenesis has been considered briefly (page 84) the electrocardiographic features are presented below with the classification

1 Supraventricular tachycardia is characterized by paroxysms in which the auricular rate is usually between 150 and 220 and the P waves are regularly spaced and separated by intervals in each lead during which there is no electrocardiographic evidence of auricular activity In order to establish the diagnosis rapid regularly spaced ectopic P waves should be identified These P waves should be separated by intervals devoid of auricular activity in all leads This is important in the differentiation from auricular flutter in which continuous regular auricular undulations are recorded in at least part of the leads The P waves may be obscured in some or all of the routine leads by superimposition on the T wave but are usually detectable in HV_1 If not they are likely to be discernible in esophageal leads at the atrial level

The onset and/or termination may be recorded thereby confirming the ectopic source of the P waves associated with the tachycardia At the onset a supraventricular premature beat initiates the succession comprising the ectopic tachycardia The episode terminates with a pause sometimes followed by one or more escaped beats from another focus before resumption of sinus rhythm If spontaneous termination is not recorded the episode may be stopped by carotid sinus pressure reflex or pharmacologic vagal stimulation which should be performed while a lead exhibiting P waves is being taken Supraventricular tachycardia is subdivided in accordance with

a The form of the P wave—2 varieties are distinguishable

1) Auric [illegible] of supraventricular tachycardia [illegible] rhythm and bear close resem- [illegible] P waves may be discernible at first glance because of fusion with the T waves under these circumstances careful scrutiny should be

directed especially towards Lead HV_1 to detect separate P and T waves Esophageal leads at the atrial level may be needed P-R interval is 12 sec or greater and is often prolonged

2) Nodal tachycadria The direction and form of the P wave are similar to those in passive A-V nodal rhythm and rate may range down to as low as 100 The P waves may precede the QRS by an interval of less than 12 sec may be superimposed upon the QRS or may fall between the QRS and T depending upon the point of origin in the auriculoventricular node and the comparative speeds of retrograde conduction through the A-V node and auricles and antegrade conduction to the ventricles

b The nature of the ventricular response during the paroxysm The ventricles may respond to each auricular impulse (1 1 ratio) to every other impulse (2 1 ratio) or irregularly (variable block), rarely the ventricles may beat at a slow independent rhythm (complete A-V block) The QRS complexes are often normal in duration and contour (i e supraventricular in form) but may be broad and/or bizarre in contour * due either to aberrant conduction through normal ventricles or to a complicating ventricular lesion causing defective intraventricular conduction The distinction between the latter possibilities may not be possible until the restoration of sinus rhythm Upon resumption of sinus rhythm after cessation of the ectopic tachycardia the QRS-T complexes may be identical with those before the paroxysm or may show significant changes referable to subepicardial or subendocardial injury etc

2 Auricular flutter Auricular activity is manifested by uniform undulations (F waves) that are continuous and without isoelectric interludes in at least some of the leads, but not necessarily in all leads The auricular rate is generally between 250 and 360 with extremes of 200 to 400 The undulations are usually best developed in Lead aV_F † sometimes in a right precordial lead (HV_1 V_{3R} or V_1) Even though one or more of these leads appears to show discrete P waves, separated by interludes devoid of auricular activity, the diagnosis of flutter can be established from the presence of continuous uniform auricular undulations in at least one lead The undulations are regular in spacing‡ and are constant in amplitude and contour in any given lead provided that allowance is made for the distortion produced by the superimposed QRS and T waves The diphasic form of the undulations often bears a resemblance to saw teeth but sometimes only the troughs or crests of the waves can be made out because of obliteration of the remainder of the undulation by superimposed QRS and T waves In the rare instances when onset of auricular flutter has been recorded the paroxysm begins with an auricular premature beat

The ventricles most commonly respond regularly to each second or fourth undulation (2 1 or 4 1 ratio) Brief episodes of 1 1 response may occur A 2 1 flutter is often converted to a higher ratio while carotid sinus pressure is applied but reverts to the original status as soon as the pressure is relinquished Rarely a regular ventricular rhythm may be maintained at some other ratio such as 3 1 If a regular ratio is present the QRS should fall consistently at the same phase of the auricular undulation

The QRS-T is usually not altered with 4 1 ratios but may become aberrant with 2 1 ratios or faster ventricular rates The aberrance is differentiated from intrinsic QRS-T abnormalities by its disappearance after significant ventricular slowing A slow but perfectly regular ventricular rhythm mathematically unrelated to the auricular rate and manifested by QRS complexes falling at different points on the undulations indicates a

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*Ventricular tachycardia characterized by rapid bizarre QRS T complexes is differentiated by the recognition of independent or retrograde P waves (page 114)

†When the undulations are not evident at a glance the presence may be suspected from the recording of bizarre auricular troughs in Lead aV_F and should lead to careful measurement

‡Measurements of consecutive auricular cycles have shown variations of less than 01 sec in duration

complete A V block Irregular spacing of the QRS is a common ventricular response to auricular flutter and results in variable ratios which usually fluctuate between 2 1 and 4 1 When the ratio is variable the QRS may fail at different points on the flutter undulations

-- undulations that are totally irregular
ally irregular ventricular rhythm

a <u>Auricular activity</u> In place of discrete P waves there are rapid undulations of varying size contour and spacing occurring at rates generally between 400 and 600 accompanied by a totally irregular ventricular response The amplitude of the auricular undulations in fibrillation is subject to variation not only in different patients but also in different leads of the same individual and has led to classification into two overlapping subgroups coarse and fine fibrillation The voltage may be so low in limb and left precordial leads that evidence of auricular activity is not discernible Under these circumstances the characteristic undulations of auricular fibrillation can usually be made out in V_{3R} V_1 HV_1 or esophageal leads at the atrial level If evidence of auricular activity is not detectable the diagnosis of auricular fibrillation may be reached inferentially from the total irregularity in the spacing of QRS complexes of supraventricular origin

Auricular fibrillation is usually differentiated readily from flutter by the marked irregularity of the auricular undulations in the former and the perfect regularity in the latter however transitional forms are observed in which the undulations are almost but not exactly, regular and uniform and are referred to as impure flutter or flutter fibrillation Undulations due to somatic tremor are easily distinguished by their sharper and more jagged contour An underlying regular sinus rhythm can be established in the presence of tremor by the demonstration of discrete P waves in at least some of the cycles and by the regular ventricular response The very rare diaphragmatic flutter is manifested by more uniform undulations, but an underlying independent normal cardiac rhythm is recognized by regular superimposition of P and QRS T complexes Undulations in the string shadow resembling those of fibrillation may occur in records made with the string galvanometer when high skin resistance has not been broken down by proper application of electrode jelly, this type of artefact should give no trouble if tracings showing standardization curves requiring more than 02 sec are rejected

b <u>Ventricular activity</u> in response to auricular fibrillation is characterized by total irregularity in spacing of the QRS-T complexes The totally irregular ventricular response is subject to *great variations* in rate depending upon the degree of functional block in the A V node and may be as high as 200 to as low as 50 An exception to the totally irregular ventricular rhythm occurs in the presence of a complicating complete A V block the existence of auricular fibrillation is established by the irregular auricular undulations and the complete A-V block by the perfectly regular ventricular rhythm

When all of the QRS-T complexes are derived from impulses transmitted through the A V node they are usually but not necessarily uniform in shape in any given lead An impulse transmitted from the fibrillating auricles through the A-V node may reach the ventricles before they have fully recovered from the previous beat and consequently may spread through the ventricles in an aberrant fashion to give rise to a bizarre QRS T complex following closely upon a normal QRS-T and resembling a ventricular premature beat The differentiation between auricular fibrillation with intermittent aberrant intraventricular conduction and that accompanied by ectopic ventricular beats is difficult unless the bizarre QRS-T complexes are frequent If of ectopic ventricular origin they will show a constant temporal relation to the antecedent ventricular complex, if due to aberrant intraventricular conduction

of a supraventricular impulse the relation to the antecedent ventricular complex will be variable A definite diagnosis of auricular fibrillation complicated by premature ventricular beats is justified when there is fixed coupling Impulses from an ectopic ventricular focus may be conducted backward into the A-V node and make this structure refractory to downcoming impulses from the fibrillating auricles to cause a more or less uniform lengthening of the time interval between the ectopic and the next supraventricular beat

IV ECTOPIC VENTRICULAR FOCUS may coexist with the sinus or auricular pacemaker, in which event the latter controls the auricles the former the ventricles Rarely the ventricular pacemaker may usurp control of the entire heart by retrograde activation of the auricles

A COEXISTENT SINUS (OR AURICULAR) PACEMAKER AND ECTOPIC VENTRICULAR FOCUS OR PACEMAKER The sinus and ventricular pacemakers may be concurrent or consecutive, as determined from a study of the entire tracing Cycles with concurrent pacemakers show evidence of independent auricular and ventricular activity Tracings with consecutive pacemakers show a series of beats in which the whole heart is controlled by supraventricular impulses interspersed with episodes in which the ventricles are controlled by an ectopic ventricular focus and the auricles by a supraventricular focus

1 Concurrent supraventricular and ventricular pacemakers *Falling* into this category, there are certain arrhythmias which were more appropriately discussed above under another classification and certain arrhythmias that belong primarily under this heading

a Supraventricular pacemaker with (premature or escaped) ectopic beats — discussed on page 106

b Ventricular parasystole, complicating sinus rhythm, is suspected when ectopic complexes that are of uniform contour in any given lead, like premature beats from a single focus fall at different points in relation to the QRS-T complexes of sinus origin Premature beats from a single focus should bear a fixed relationship to the preceding cycle The presence of a ventricular parasystolic rhythm is verified by demonstrating that *the interval between any two ectopic complexes that are separated* by cycles of sinus origin is an exact multiple of the interval between two contiguous ectopic complexes

c Complete A-V block with ventricular activation from an idioventricular pacemaker discussed on page 97

d Escaped ventricular rhythm with retrograde block and interference dissociation This is a rare variant of an escaped nodal mechanism with interference dissociation discussed on page 109 and differs merely in the site of the pacemaker for the ventricles The decision as to whether the *lower pacemaker lies in the A-V node* or ventricles is made from the contour of the QRS-T during the escaped rhythm as compared with that during sinus control QRS complexes like those during sinus control indicate a nodal pacemaker *broad and bizarre QRS complexes point to a* ventricular pacemaker

e Ventricular tachycardia with retrograde block is an active ectopic tachycardia generally above 170 but ranging from 120 to 250 and characterized by broad bizarre QRS complexes with oppositely directed T waves completely independent of all antegrade impulses arising above the bifurcation of the bundle of His as shown by the fact that the P waves fall at different points *in reference to the QRS T and* occur at a rate mathematically unrelated to the ventricular rate Ventricular tachycardia is differentiated from auricular tachycardia complicated by bundle branch block by demonstrating *an independent* auricular rhythm with P waves falling at variable points in reference to the QRS-T

Ventricular tachycardia occurs in short or long paroxysms which begin abruptly with a ventricular premature beat and terminate with a pause followed by resumption of sinus (or nodal) rhythm The ventricular ectopic beat that appears to initiate a paroxysm of tachycardia is prone to be very premature and superimposed upon the *T wave* of the preceding cycle During the paroxysm, the ventricular rhythm may be perfectly regular or may show slight irregularities *in cycle length*

The QRS complexes are prolonged in duration and coarsely slurred or notched, the T waves are readily demarcated and are opposite in direction to the main deflection of the QRS The approximate location of the focus is determined from the QRS patterns in multiple precordial leads A QS complex is recorded in the lead *or leads* overlying the epicardial surface of the ventricular segment from which the tachycardia is originating a monophasic R wave is obtained in leads over the *opposite wall of the* heart a diphasic RS complex is registered in intervening leads The upright component of this RS complex is small and brief in duration in leads over portions of the ventricle near the ectopic focus and increases in amplitude and duration at the expense of the S wave in leads farther and farther *removed* from the focus,

Multifocal ventricular tachycardia is characterized by ectopic QRS-T *complexes of* two or more distinctive types *in a given lead* *Each type of QRS* complex is *independent of auricular activity* and is prolonged in duration coarsely slurred or notched and *followed by a readily* demarcated T wave of opposite direction The *most common finding is* an alternation between two different types of QRS-T patterns the so called bifocal ventricular *tachycardia*

f <u>Ventricular flutter</u> *In place of* a clearly definable QRS *complex and* T wave, the ventricular deflections consist *of rapid undulations (i e over 250* per *minute)* in which QRS and T components are *blended and indistinguishable* Ventricular flutter is differentiated from *ventricular fibrillation by the* fact that the undulations are regular and uniform in the *former irregular in spacing and variable* in contour and amplitude in the latter If *auricular activity is discernible in the tracing* the P waves are independent of the *ventricular undulations*

2 *<u>Consecutive supraventricular and ventricular</u>* <u>pacemaker</u> is a rare variant of the arrhythmia wandering pacemaker *between the* sinus and A-V *nodes*, described on page 110 In this condition *wandering pacemaker* between the sinus *node* and ventricles there is a slow rate with shift from episodes *of sinus origin to episodes* characterized by QRS T complexes from a ventricular *focus* and retrograde P waves

B ECTOPIC VENTRICULAR PACEMAKER *replacing* the sinus pacemaker, is *characterized by regularly* spaced bizarre *QRS T complexes with oppositely* directed T waves and by evidence of conduction backward into the *auricles namely*, retrograde *P waves following* each *QRS or* after every second *third* or fourth ventricular complex When tracings with an ectopic ventricular *pacemaker fail* to show evidence *of auricular activity* in any lead, including RV_1 the *auricles may be* at standstill and the case may be *classifiable* in a category of single cardiac *pacemaker* Irrespective as to whether *there* are retrograde P waves or auricular standstill *the* ectopic ventricular *rhythm may be classified* according to rate either as passive (below *100) or active*

1 Pass[illegible]

with retrograde *P waves or* auricular standstill

2 [illegible] 14) *may be* complicated by auricular standstill or the latter manifested by *P* waves following the QRS and inverted in aV_F upright in aV_R

3 <u>Ventricular flutter</u> (page 115) *P waves are usually* not discernible

4 <u>Ventricular fibrillation</u> *is characterized* by irregular undulations of varying size and shape without demarcation between QRS and T P waves are usually not discernible In records that include *the onset* ventricular fibrillation is heralded by *one or* more premature ectopic ventricular beats

E. SUMMARY OF FINDINGS IN UNIPOLAR THORACIC AND LIMB LEADS

NORMAL ADULT

I THORACIC LEADS

A QRS INTERVAL — usual range 06 to 10 sec with average of 08 sec Measurements of 11 sec have been obtained rarely in apparently normal individuals, but should not be passed over as a normal variation unless the remaining electrocardiographic and clinical findings are normal in every respect

B Q-T INTERVAL varies with sex and cardiac rate The decision as to whether or not the Q-T interval in a given case is normal is best reached by reference to the table of normal standards of Ashman and Hull The following close approximation of their values is easy to remember and will serve when their table is not available

Heart Rate	60	70	80	90	100	120
Upper limits of normal for men	42 sec	40	38	36	34	31
Average normal values for men	39 sec	37	35	33	31	28
Corresponding values for women	01 sec longer than those for men					

For cardiac rates between 60 and 100 per minute the Q-T interval decreases by 02 sec for each increase of 10 beats per minute in cardiac rate The upper limits of normal are approximately 03 sec greater than the average values

C TRANSITIONAL ZONE In a series of leads across the front of the chest at the ventricular level and in a comparable series across the back of the chest contrasting patterns should be present in leads from the right and left ends of each series and a transition should be demonstrable in leads from intervening points This transitional zone corresponds roughly to the area of reference of the potential variations of the anterior and posterior ends of the interventricular septum Since leads from the right side of the back are not taken as a routine, the posterior transitional zone is usually not demarcated The following remarks apply to the localization of the anterior transitional zone which is essential to the delineation of the zones of reference of the potential variations of the respective epicardial surfaces of the right and left ventricles

1 Location is subject to variation, depending upon the degree of rotation of the heart on its 3 axes particularly the longitudinal axis The usual location is somewhere between chest positions 2 and 4, so that Lead V_2 and leads from the right precordium reflect principally the potential variations of the epicardial surface of the right ventricle and Lead V_4, and leads farther to the left reflect chiefly the potential variations of the epicardial surface of the left ventricle *Clockwise rotation of* the heart tends to displace the *transition* zone leftward as far as chest position 5 and rarely to position 6 *Counterclockwise* rotation may displace the transitional zone to the right between positions 1 and 2

2 Width of transitional zone is subject to considerable variation, probably dependent upon the anatomic relations of the long axis of the septum to that of the precordial electrode positions

a Abrupt transition is found when these 2 axes are approximately perpendicular to one another and is characterized by a striking contrast in the QRS patterns in 2 adjacent leads The nature of the abrupt change in the QRS depends upon whether the electrode moves from a point over the right ventricle across the septum to a point over the left ventricle in 2 adjacent chest leads or whether it moves from a right ventricular position to a point directly over the septum and then to a left ventricular position in 3 consecutive leads In the former there is an abrupt change from an rS pattern to an Rs pattern in adjacent leads * In the latter a *transitional* rs complex is recorded characterized by (1) an upstroke and a downstroke of approximately equal amplitude (2) total voltage measured from peak of r to nadir of s significantly lower than in the 2 adjacent leads, (3) frequency of notching or coarse slurring

b Gradual transition is found when the 2 axes are more parallel to one another Under these circumstances transitional complexes are recorded in 2 or more leads

D QRS-T PATTERNS IN RIGHT VENTRICULAR LEADS AS CONTRASTED WITH THOSE IN LEFT VENTRICULAR LEADS

RIGHT VENTRICULAR LEADS	LEFT VENTRICULAR LEADS
QRS Direction of initial deflection	
1 Characteristically upright in all *leads over* right ventricle due to *initiation* of activation of septum in left to right direction	1 Upright in leads just to the left of the transitional zone and produced by early activation of anteroseptal wall of left ventricle *Downward* in leads farther to the left due to septal activation from left to right prior to the arrival of the impulse in subendocardial aspect of lateral and posterior *walls* To be considered normal the q wave in all left ventricular leads must conform to the following requirements a duration from onset to nadir — 02 sec or less b amplitude less than 25% of the succeeding R
2 As a variant a QS *complex* may be recorded Limitation to leads over	2 As a normal variant, an initial R wave may be recorded in all customary leads

*The lower case letter is used to indicate a relatively small deflection the upper case letter to indicate a relatively large deflection

the right atrium (with an rS in leads over the right ventricle is a normal finding, observed when the relatively small potentials from septal activation disappear with distance and recognized by the fact that the resultant QS is briefer than the rS recorded in leads farther to the left The registration of a QS complex in leads over the right ventricle as well as the atrium, may be a normal variant in which event a normal initial upstroke will be recorded in all leads over the apical portion of the left ventricle as described *in the right-hand column* These findings are presumably an *expression of initiation of activation* of the apical portion of the septum by impulses which enter from the right Purkinje network and penetrate towards the left — an alternative of *the left to right vector usually found* in normals

facing the apex of the left ventricle including V_7 and V_8*, it is characterized by a smooth upstroke consuming less than 04 sec from onset to peak and a total QRS duration below 10 sec Incomplete left bundle branch block is also accompanied by a right to left septal vector but is distinguished electrocardiographically by a slurred or notched initial upstroke consuming more than 04 sec from onset to peak and a total QRS duration *that is usually between 10* sec and 12 sec Even though the findings in all leads facing the apex of the left ventricle exhibit a normal initial R wave from a normal right to left vector *in the apex of the left ventricle HV leads* facing the left side of the base of the septum should display a normal q wave since the base of the septum is always activated normally after the apex and in *a left to right vector The normal* q wave in HV leads facing the left ventricle should be less than 03 sec in duration and less than 25% of the amplitude of the succeeding R wave

R/S relationships and contour

1 The *characteristic finding in* all leads to the right of the transitional zone is an rS complex The small r wave normally recorded in leads over the tricuspid ring reflects the sum of positive potentials reaching the precordium from activation of (a) the septum in the usual left to right direction, (b) the free wall of the right ventricle, minus the opposing potentials coming from activation of the lateral and posterior walls *of the left ventricle* the large S wave is derived from continuing activation of these portions of the left ventricle after completion of septal and right ventricular depolarization *As the electrode is* moved over the right ventricle towards the transitional zone the r wave increases in amplitude and duration, due to the increasing

1 *The characteristic finding in the lead or* leads just to the left of the transitional zone is an Rs complex The relatively large R wave in these leads is produced mainly by activation of the anteroseptal wall of the left ventricle with a comparatively small contribution from the anteroseptal wall of the right ventricle and represents the sum of these forces minus the opposing potentials derived from the posterior wall The continuing activation of the posterior wall after arrival of the impulse at the epicardial surface of the anteroseptal wall is responsible for the relatively small S wave usually present in leads just to the left of the transitional zone Slight notching or slurring near the base of the ascending limb of the R wave occurring as part of an Rs complex recorded just to the left of the transitional zone may be observed as

*An initial deflection that is downward only in Lead V_8 or in Leads V_8 and V_7 indicates the presence of the usual left to right septal vector the registration of an initial R wave in V_6 and V_5 under these circumstances is merely a manifestation of early activation of the anterolateral wall of the left ventricle

contribution of positive potentials transmitted from the epicardial surface of the anteroseptal wall of the left ventricle The S wave attains its greatest depth in V_1 or V_2 and diminishes as the electrode approaches the left ventricle

2 As a common normal variant slurring or fine notching of the ascending or descending limb of the r wave may be observed due to im-
[illegible]

3 As a rare normal variant an rSr' complex may be recorded in Leads V_1 V_2 or HV_2 The r' deflection may be within normal limits if it does not exceed 3 mm in amplitude It is believed to be a manifestation of the normal late activation of the crista supraventricularis a thick muscular ridge in the roof of the right ventricle between the atrioventricular and pulmonary orifices In the absence of hypertrophy of this structure or other portions of the right ventricle the forces developed during its late activation are seldom sufficient to be registered in precordial leads, but occasionally may cause an r' deflection up to 3 mm in height It has been claimed that an r' deflection may be recorded in Lead V_1 as a result of transmission of the potential variations of the posterior wall of the left ventricle to the right sternal border but convincing evidence has not been obtained Although transmission of these potentials across the back to the right arm has been demonstrated transmission anteriorly to the right sternal border would require extreme clockwise rotation difficult to reconcile anatomically Such a possibility could not even be considered unless there was a pronounced late R wave in aV_R but then might have to be investigated by Leads V_{4R} to V_{9R} inclusive

a normal variant produced by potentials derived from activation of the adjacent anteroseptal wall of the right ventricle As the electrode is moved farther to the left a q wave appears due to later arrival of the impulse in the lateral and posterior walls and the s wave disappears, due to the resultant late completion of activation of these areas When a qR or qRs complex is present the ascending limb of the R wave is normally smooth and should not exhibit definite notching or coarse slurring This R wave reflects the forces created by activation of the lateral or posterior wall of the left ventricle, transmitted from its surface minus any opposing forces transmitted from the opposite anterolateral wall of the right ventricle The latter are probably negligible under normal circumstances, but reach significant proportions in right ventricular hypertrophy and conduction defects

Time from onset of QRS to peak of R (i e onset of intrinsicoid deflection) in a representative lead By comparison of the measurements in right with

left ventricular leads, one determines which ventricle is activated first and which last By consideration of the actual figures one determines whether the time consumed in activation is normal or prolonged

This measurement includes the time for activation of the septum in the usual *left to right direction plus the time for* activation of the free wall of the right ventricle and is preferably made in leads *overlying the tricuspid ring so* as to minimize effects from the anteroseptal wall of the left ventricle, the normal finding is less than 03 *sec* and reflects the early and rapid activation of the relatively thin right ventricle

This is an approximation of the time elapsing between the onset of septal activation and the arrival of the impulse at the epicardial surface of the subjacent outer wall of the left ventricle The *normal finding* is less than 06 sec and reflects the later onset and completion of activation in the left ventricle As the *electrode is moved from positions over* the right to positions over the left ventricle this measurement progressively lengthens to a maximum reflecting greater and greater contribution from the left ventricle In leads with a q wave the time required for passage of the impulse from the subjacent endocardium to epicardium is approximated by the time elapsing from onset to peak of the R wave The normal duration of the upstroke of the R wave is less than 04 sec in the lead with the longest measurement

S-T junction and segment

1 Isoelectric S-T junction is the typical normal finding This means that the S-T junction is at the same level as the T-P segment

2 Elevation of the S-T junction to the extent of 5 to 2 0 mm and rarely 3 0 mm may occur as a normal variant in the presence of tall upright T waves particularly in leads over the right ventricle or in leads at or just to the left of the transitional zone Presumably this is due to the relatively large electromotive force already developed by the repolarization process at the time of the termination of the QRS An elevation of the S-T junction may be considered normal if the displacement does not exceed 3 mm and provided the S-T segment describes an arc with upward concavity ending in a tall upright T wave *On the other hand, lesser elevations of the S-T junction (1 0-1 5 mm)* may be significant if the S-T segment is abnormal in contour

3 Depression of the S-T junction in precordial leads is rare and should be regarded as abnormal when it amounts to 5 mm or more provided the following types of pseudodepression are excluded

a Apparent depression may occur in the presence of a tachycardia when the normal isoelectric T-P segment is not recorded due to the fact that the P wave begins prior to the completion of the T The only recourse is to judge the position of the S-T junction in reference to the T_P junction with the beginning of QRS

b A prominent auricular T wave causing a depression of the S-T junction is recognized from the presence of a tall P wave and a reciprocal depression of the interval between the end of the P wave and the beginning of the QRS When the correct allowance cannot be made for the T_P wave in judging the position of the S-T junction, the measurement should be made with reference to the P-R junction rather

than the T P *segment* When there is pseudodepression of the S-T junction due to tachycardia and/or a prominent Tp wave the S-T segment maintains the normal *contour of a progressive* ascent in an upwardly concave arc When there *is* abnormal depression of the S-T *junction* due to subendocardial injury the segment pursues an appreciable horizontal course or sags downwards The contour of the S-T *segment* thus aids greatly in distinguishing between pseudodepression and abnormal depression from subendocardial injury

4 The contour of the S-T segment may be classified as straight, *convex upward, or concave upward* and is normally related to the direction of the T wave When the T is upright the S-T segment characteristically slopes gently *upward to the peak of the* T in an arc with upward concavity, when the T wave is inverted a gently sloping arc is directed downward with upward convexity *Loss of this characteristic curvature may* be within normal limits when confined to leads at the transitional zone or in any lead *in the presence* of tachycardia The contour of the S-T segment should be regarded as abnormal when a straight slope is present under other circumstances or when the convexity points in the same direction as the T wave A *very* slight downward sagging or an undue horizontal course of the S-T segment should arouse suspicion and should prompt an exercise test unless there are clinical contraindications (page 143)

T wave

Right ventricular leads

1 Direction of the T wave in leads over the right atrium is subject to the following normal variations

a Upright when the electrode is reflecting chiefly the potential variations of the epicardial surface of the right ventricle

b Inverted when the electrode is reflecting chiefly the potential variations of the ventricular cavities The normally inverted T wave seldom exceeds 3 mm in depth

c Diphasic or flattened with varying mixtures of surface and cavity potentials

2 [illegible]

in normal children In the latter the the inversion is *greatest* in leads over the right atrium or atrioventricular groove and recedes in *progressively* leftward electrode *positions* The T wave may still be negative in leads at the transitional zone but becomes upright over the left *ventricle* Occasionally this juvenile pattern persists into the third decade especially in women

Left ventricular leads

1 [illegible] of T wave in leads over the [illegible] are [illegible] over the *left ventricle* but the following exceptions may be observed as normal variants

a Flattening may occur in axillary and back leads in association with low voltage *of the* R *wave* (below 5 mm) and may be considered as a normal variant if no abnormalities in QRS-T pattern are found throughout the tracing

b Inversion of the T wave may occur in posterior leads near the *atrioventricular* groove (HV_8 HV_9 and V_9)



3 Amplitude Upright T waves in precordial leads may range from 1 to 12 mm in height Before classing a T wave in the upper part of this range as a normal variant reciprocal exaggeration in amplitude secondary to injury of the opposite wall of the ventricle must be excluded For example, when T waves are tall in precordial leads over the right ventricle and anteroseptal wall of the left ventricle, care must be taken to exclude posterolateral infarction

4 Contour Normal upright T waves are characteristically smooth in contour with upward concavity of both the proximal and distal limbs resulting in a blunt to moderately sharp peak The slope of the distal limb is usually steeper than the gradient of the proximal limb Notching or double peaking may occur as a normal variant in leads at the transitional zone This should be distinguished from pseudodoubling occurring when the U wave is prominent

II UNIPOLAR LIMB LEADS

A LEAD aV_R The normal variations encountered in this lead are dependent primarily upon the direction of rotation of the heart on its transverse axis

1 Forward rotation of the apex Under these circumstances the portion of the heart directed towards the right arm consists largely of the atria and great vessels and includes little or none of the epicardial surface of either ventricle As a consequence the major deflections of ventricular origin are derived from the potential variations of the endocardium and cavities of the ventricles and consist of a downward QRS and inverted T wave This basic pattern is normally subject to 4 minor variations namely a monophasic QS a minute r followed by a deep S, a deep Q followed by a minute r and an rSr' complex When any of the foregoing QRS patterns is present the chief interest lies in the S-T segment and T wave The S-T junction is normally isoelectric the segment curved with upward convexity and the T wave inverted

2 Backward rotation of the apex facilitates transmission of the potential variations of the epicardial surface of the posterobasal wall of the left ventricle to the right arm as well as those of the ventricular cavities, and is manifested by the registration of a QR complex characterized by a late R wave ranging from 33% to 100% of the amplitude of the antecedent Q wave This QR complex must be differentiated from the abnormal rsR' or sR' wave recorded in Lead aV_R as a result of right ventricular hypertrophy or right bundle branch block The S-T junction is normally isoelectric and T wave inverted

B LEADS aV_L and aV_F taken together reflect the direction and degree of rotation on an anteroposterior and a longitudinal axis This type of rotation is influenced particularly by the height of the diaphragm and the shape of the chest Elevation of the diaphragm causes counterclockwise rotation into a horizontal or semihorizontal position In either position the potential variations of the left arm are dominated by those of the left ventricle in the former the potential variations of the left leg are transmitted chiefly from the right ventricle and in the latter chiefly from the posterior transitional zone and adjoining walls of the two ventricles An intermediate position of the diaphragm permits reference of the potential variations of the left ventricle to both extremities Lowering of the diaphragm facilitates clockwise rotation into a semivertical or vertical position In either position the potential variations of the left leg are dominated by those of the left ventricle in the former the potential variations of the left arm are derived chiefly from the anterior transitional zone in the latter from either the epicardial surface of the right ventricle or left atrium depending upon the degree of rotation on the longitudinal axis The normal variations in P waves (assuming sinus rhythm) and in QRS-T pattern in Leads aV_L and aV_F classified according to electrical position are summarized in tabular form

LEAD aV_L

HORIZONTAL – SEMIHORIZONTAL – INTERMEDIATE

1 Transmission predominantly from epicardial surface of lateral wall of the left ventricle

a P wave upright

b QRS resembling that in precordial or axillary leads over left ventricle and judged as normal or abnormal by same criteria as applied to corresponding chest leads

1) Rs or monophasic R Ascending limb of R less than 04 sec in duration

2) qRs or qR The q should not exceed 02 sec in duration or 25% of amplitude of the succeeding R

c T wave upright but as a normal variant may be flattened if R wave less than 5 mm

2 Transmission from posterobasal aspect of left ventricle and to a variable extent from ventricular cavities through atrioventricular orifices

a P wave inverted or diphasic

b QRS resembling that in high back leads (HV_8) and characterized by qRs or qR pattern Owing to possible derivation from cavity potentials transmitted through AV orifice q should not be considered abnormal unless 04 *sec or more* in duration

c T wave usually upright but may be flat or shallowly *inverted*

LEAD aV_F

P wave upright in all electrical positions

HORIZONTAL

1 Transmission predominantly from epicardial aspect of posterior inferior wall of right ventricle

a QRS resembling that in precordial leads over the right ventricle An rS complex is the usual normal finding, a QS is an occasional normal variant Since the QS generally results from loss of the initially positive potentials in the course of transmission from the surface of the right ventricle to the leg the total QRS interval under such circumstances is significantly shorter than in precordial leads Often there is sufficient respiratory shift in diaphragmatic position to result in phasic variation from a normal QS to rS complex but not to a Qr deflection These features distinguish the normal QS from the abnormal QS resulting from transmural posterior infarction In the latter a small late r is usually detectable (page 153)

b T wave most commonly upright, but may be normally flattened diphasic or inverted as in V_1

SEMIHORIZONTAL

1 Transmission predominantly from posterior end of septum and adjoining posteroseptal aspects of right and left ventricles

a QRS consists of either a multiphasic complex of low voltage or an *rs* deflection that is approximately equiphasic and usually low in voltage

b T wave usually upright but may be flattened

INTERMEDIATE

1 Transmission predominantly from posteroapical surface of left ventricle QRS and T patterns are described below

LEAD aV_L

SEMIVERTICAL

1 Transmission predominantly from anterior end of septum and adjoining anteroseptal aspects of right and left ventricles
- a P wave may be upright diphasic, or inverted
- b QRS consists of either a multiphasic complex of low voltage or an *rs* deflection that is approximately equiphasic and usually low in voltage
- c T wave usually upright but may be flattened

VERTICAL

1 *Transmission predominantly from* epicardial surface of right ventricle
- a P wave may be upright, flat diphasic, or inverted
- b QRS resembling that in precordial leads *over the right ventricle An* rS complex is the typical normal finding, *r*Sr' or QS complexes constitute occasional normal variants If the rSr' is within normal limits the r' component should be small (2 mm or less) and should be traceable to a normal r' in right precordial leads, presumably derived from the crista supraventricularis The QS complex generally results from loss of the initially positive potentials in *course of transmission* from the surface of ventricle to the left arm hence the total QRS interval is significantly shorter than in precordial leads This together with the usual accompanying P wave inversion, aids in distinguishing the normal QS from the abnormal *associated with* lateral infarction
- c T wave may be upright flat, diphasic or inverted

2 Transmission predominantly from ventricular cavities through atrioventricular valves
- a P wave inverted or diphasic
- b QRS resembling that in high *esophageal leads and* characterized by a Qr complex The suspicion

LEAD aV_F

SEMIVERTICAL AND VERTICAL

1 Transmission predominantly from posteroapical aspect of left ventricle
- *a* P wave *upright with* gently sloping limbs
- b QRS resembles that in leads from the stomach and from the back below the diaphragm In normals and in the absence of *localized left* ventricular disease the QRS pattern in aV_F of an intermediate semivertical or vertical heart resembles that in precordial and axillary leads over the left ventricle It is judged as normal or abnormal by essentially the same criteria *applied to the corresponding chest* leads
  - 1) Rs or monophasic R The ascending limb of the R is less than 04 sec in duration
  - *2) qRs or qR The q should be less* than 03 sec in duration and less 25% of the amplitude of the R wave provided QRS voltage is over 0 5 mv When voltage is low, Q/R ratios are undependable and decision as to whether the Q wave is normal or abnormal is based *solely on time from its onset to* nadir
- c T wave upright but as a normal variant may be flattened if R wave is less than 5 mm

2 Transmission predominantly from posterobasal aspect of left ventricle This may occur when diaphragm is low and *cardiac apex is displaced forward* This position is suspected from the contour of the P wave
- a P wave tall upright with a precipitously descending terminal limb representative of the intrinsicoid deflection found in esophageal leads near the atrioventricular groove
- *b QRS consists of a qR or qRs pattern* generally of relatively high voltage The q should be less than 03 sec in duration less than 25% of the amplitude of the R

LEAD aV_L	LEAD aV_F
that the Qr complex is a normal the presence of an inverted or diphasic P wave The establishment of the Qr as a normal variant is further aided by finding drastic changes in the relative amplitude of the downward and upright components with shift in posture However the final decision as to presence or absence of infarction of the base of lateral or posterior wall is made from the pattern in high chest leads c T wave inverted	c T wave usually upright but as a normal variant may be flat diphasic or shallowly inverted

BUNDLE BRANCH BLOCK

A method for the analysis of tracings with prolonged or abnormally slurred or notched QRS complexes has been given on pages 25 - 27 As a prerequisite to the establishment of an intraventricular conduction defect it is necessary to exclude an ectopic ventricular focus (as in ventricular tachycardia idioventricular rhythm etc) and anomalous ventricular excitation as in the Wolff-Parkinson-White syndrome If a study of the rhythm indicates that the prolonged bizarre QRS complexes resulted from impulses of supraventricular origin that reached the ventricles through the A-V node the ventricle exhibiting the conduction defect is determined from the location of the chest leads exhibiting an abnormally late onset of the intrinsicoid deflection The decision as to whether the conduction defect is in the bundle branch or free wall of the ventricle is based upon the direction of the initial phase of the QRS in all leads with a late intrinsicoid deflection

I CLASSICAL LEFT BUNDLE BRANCH BLOCK (LBBB) In complete LBBB and in the classical case of incomplete LBBB the QRS complexes in the high precordial leads resemble those in the customary precordial leads in respect to the direction of the initial deflection The registration of an initial upstroke in all high as well as low left ventricular leads points to an interruption of conduction on the left side of the septum at its base with activation of the entire septum in a right to left vector The classical finding must be differentiated from a variant of incomplete left bundle branch block in which all customary leads facing the apical portion of the left ventricle (V_4-V_8 inclusive and aV_F) show an initial R wave whereas high precordial leads over the left ventricle show an initial normal q component of a qR complex This will be discussed separately The following discussion pertains to classical uncomplicated LBBB in such cases autopsy almost invariably shows left ventricular hypertrophy and may in addition disclose localized lesions in the left ventricle In almost all cases of coexisting complete LBBB and infarction of the left ventricle the broad slurred or notched initial R characteristic of LBBB is recorded in all left ventricular leads and no QRS abnormalities diagnostic of infarction are detectable With extensive infarction of the septum, causing LBBB there is a possibility of an initial Q wave in left ventricular leads, as will be discussed on page 151

A QRS INTERVAL

1 Complete LBBB — 12 sec or longer
2 Incomplete LBBB — 10 to 11 sec

B Q-T INTERVAL – characteristically beyond upper limits of normal

C LOCATION OF TRANSITIONAL ZONE Although the transitional zone may be in the usual normal location (between V_2 and V_4) it is more commonly shifted to the left presumably because of right ventricular dilatation It is often between chest positions 4 and 5, not uncommonly between 5 and 6, and occasionally between 6 and 7 In most cases, the only leads exhibiting a late intrinsicoid deflection and no S wave are V_8 $V_8 + V_7$, or $V_8 + V_7 + V_6$ and the corresponding high leads

D QRS-T CONFIGURATION IN LEADS OVER THE RIGHT SIDE OF THE HEART

1 QRS complex In the presence of left bundle branch block, the impulse arrives early in the right side of the septum and free wall of the right ventricle and late in the free wall of the left ventricle The late activation of the outer wall of the left ventricle is always manifested by a deep, broad, slurred S wave in leads facing the epicardial surface of the right ventricle and atrium The initial phase of the QRS complex in these leads depends upon the interplay of negative potentials, transmitted from the endocardial surface of the right side of the septum and positive potentials produced by activation of the free wall of the right ventricle As a consequence the QRS pattern in any given lead over the right atrium or ventricle may take one of three forms, whereas the QRS interrelations in different right precordial leads are subject to considerable variation

a Varieties of QRS pattern that may be found in leads over the right side of the heart in uncomplicated LBBB

1) An rS complex consisting of a small narrow initial r wave due to early preponderance of positive potentials coming from activation of the free wall of the right ventricle and a deep, broad, slurred S wave due to activation of the septum and the free wall of the left ventricle This r wave is abbreviated by the opposing negative potentials of septal origin

2) A triphasic qrS complex the q wave representing initial negativity of the right ventricular cavity from septal activation, the r wave reflecting depolarization of the free wall of the right ventricle

3) A deep, broad, slurred QS deflection The r wave may be submerged and obscured in this monophasic downstroke because of complete dominance of negative potentials transmitted from the endocardial surface of the right side of the septum over positive potentials coming from activation of the free wall of the the right ventricle or the r wave may be represented by an equivalent in the form of notching or coarse slurring near the onset of the QS deflection

b Variations in QRS interrelations in different right precordial leads Leads over the right atrium and ventricle in uncomplicated left bundle branch block do not necessarily show either the expected increase in the initial r wave as the electrode is moved towards the left or the uniform QS or rS pattern occurring as a recognized variant A distinct rS deflection (or rarely a qrS complex) may be recorded in Lead V_1 or V_2 and a QS deflection in one or more leads closer to the right side of the interventricular septum in the presence of uncomplicated left bundle branch block and may lead to an erroneous interpretation of anterior infarction The distinct R wave in Lead V_1 and/or V_2 may be correlated with an electrode position over the tricuspid orifice favorable to the reception of positive potentials coming from the relatively thick base of the right ventricle and perhaps from the left ventricular cavity via the left and right atria whereas the decrease or disappearance of the R wave in V_3 and V_4 may be correlated with an electrode position over the relatively thin apex of the right ventricle and nearer the right side of the septum

2 S T pattern The S-T junction is always elevated and often the displacement is significantly beyond the upper limits of normal (i e greater than 3 mm) The S-T segment usually displays upward concavity but may show straightening or upward convexity particularly if digitalis had been given The T wave is characteristically tall upright

E QRS T CONFIGURATION IN LEFT VENTRICULAR LEADS

1 QRS pattern Leads just beyond the transitional zone may show an Rs pattern but those farther to the left show the classical broad slurred or notched monophasic R wave This is a composite deflection consisting of an initial upstroke or R wave proper derived from activation of the septum in a right to left direction followed by a second upstroke or R' wave derived from activation of the free wall of the left ventricle The two components may be fused into a smooth monophasic R wave in some left ventricular leads but are usually demarcated in other leads by the intervention of slurring notching or a downstroke (S) that crosses the isoelectric line When the two components are identifiable separately the period elapsing from the onset to the extinction of the initial R wave is an index of the time required for the impulse to pass through the septum and start activating the free wall of the left ventricle whereas the time from beginning to peak of the R' deflection is proportional to the time consumed in activation of the outer wall of the left ventricle

a Initial upright deflection is invariably present in leads taken well over the epicardial surface of the left ventricle and is the result of septal activation The duration of this initial R wave is a rough index of the time consumed in septal activation The measurement is made from the onset of the R to the onset of the R' wave in the presence of a notched or slurred upright complex If there is an S wave intervening between the R and R' the measurement is made from the onset of the R wave to the point where the string returns to the isoelectric line in the course of the downstroke of the S wave This measurement should amount to 04 sec or longer in complete left bundle branch block and is usually between 02 sec and 04 sec in incomplete left bundle branch block

b Late peak or R' wave The time from onset of the QRS to the peak of the R' (i e onset of the intrinsicoid downstroke) is an index of the time required for activation of the septum plus the free wall of the left ventricle The magnitude of the prolongation depends upon the degree of block

1) Complete LBBB – 08 sec or more after onset of QRS

2) Incomplete LBBB – 06 to 08 sec after onset of QRS

c Variations in QRS interrelations in different left precordial leads The portion of the QRS on which the notch or maximal slurring occurs in the presence of complete LBBB varies with the position of the electrode in reference to the transitional zone In leads over the anteroseptal wall it usually appears on the descending limb of the R and as the electrode is moved to the left it shifts to the peak then the ascending limb coincidentally the R' becomes more prominent This reflects increasing distance from the septum and purer recording of the potential variations of the outer wall of the left ventricle

2 S T segment and T wave are characteristically opposite in direction from the broad slurred or notched portion of the QRS (i e discordant) Thus in left ventricular leads the S T junction is usually depressed the segment curved with upward convexity and the T wave deeply inverted Occasionally the T wave is upright (i e , concordant) This should arouse the suspicion of a lesion excessively delaying repolarization of the subendocardial layer of the underlying wall but a satisfactory explanation is often not provided by pathologic examination

B Q-T INTERVAL – characteristically beyond upper limits of normal

C LOCATION OF TRANSITIONAL ZONE Although the transitional zone may be in the usual normal location (between V_2 and V_4), it is more commonly shifted to the left, presumably because of right ventricular dilatation It is often between chest positions 4 and 5, not uncommonly between 5 and 6, and occasionally between 6 and 7 In most cases the only leads exhibiting a late intrinsicoid deflection and no S wave are V_8, $V_8 + V_7$, or $V_8 + V_7 + V_6$ and the corresponding high leads

D QRS-T CONFIGURATION IN LEADS OVER THE RIGHT SIDE OF THE HEART

1 QRS complex In the presence of left bundle branch block, the impulse arrives early in the right side of the septum and free wall of the right ventricle and late in the free wall of the left ventricle The late activation of the outer wall of the left ventricle is always manifested by a deep broad slurred S wave in leads facing the epicardial surface of the right ventricle and atrium The initial phase of the QRS complex in these leads depends upon the interplay of negative potentials, transmitted from the endocardial surface of the right side of the septum and positive potentials produced by activation of the free wall of the right ventricle As a consequence, the QRS pattern in any given lead over the right atrium or ventricle may take one of three forms, whereas the QRS interrelations in different right precordial leads are subject to considerable variation

a Varieties of QRS pattern that may be found in leads over the right side of the heart in uncomplicated LBBB

1) An rS complex, consisting of a small narrow initial r wave due to early preponderance of positive potentials coming from activation of the free wall of the right ventricle and a deep broad, slurred S wave due to activation of the septum and the free wall of the left ventricle This r wave is abbreviated by the opposing negative potentials of septal origin

2) A triphasic qrS complex, the q wave representing initial negativity of the right ventricular cavity from septal activation the r wave reflecting depolarization of the free wall of the right ventricle

3) A deep broad slurred QS deflection The r wave may be submerged and obscured in this monophasic downstroke because of complete dominance of negative potentials transmitted from the endocardial surface of the right side of the septum over positive potentials coming from activation of the free wall of the the right ventricle or the r wave may be represented by an equivalent in the form of notching or coarse slurring near the onset of the QS deflection

b Variations in QRS interrelations in different right precordial leads Leads over the right atrium and ventricle in uncomplicated left bundle branch block do not necessarily show either the expected increase in the initial r wave as the electrode is moved towards the left or the uniform QS or rS pattern occurring as a recognized variant A distinct rS deflection (or rarely a qrS complex) may be recorded in Lead V_1 or V_2 and a QS deflection in one or more leads closer to the right side of the interventricular septum in the presence of uncomplicated left bundle branch block and may lead to an erroneous interpretation of anterior infarction The distinct R wave in Lead V_1 and/or V_2 may be correlated with an electrode position over the tricuspid orifice favorable to the reception of positive potentials coming from the relatively thick base of the right ventricle and perhaps from the left ventricular cavity via the left and right atria whereas the decrease or disappearance of the R wave in V_3 and V_4 may be correlated with an electrode position over the relatively thin apex of the right ventricle and nearer the right side of the septum

II. RIGHT BUNDLE BRANCH BLOCK (RBBB). The following discussion pertains to the uncomplicated form of RBBB. Its association with septal infarction will be discussed on page [illegible]. Complete RBBB may be ascribed to primary involvement of the right ventricle in only a minority of cases. The majority show primary involvement of the left ventricle. In some, no anatomic lesions can be found.

A. QRS INTERVAL

1. Complete RBBB — .12 sec. or longer.

2. Incomplete RBBB — .10 to .11 sec.

B. Q-T INTERVAL — characteristically beyond the upper limits of normal.

C. LOCATION OF TRANSITIONAL ZONE — subject to marked variations in different cases. Of particular importance are the electrocardiograms in which the transitional zone is found between positions 1 and 2 or even to the right of position 1. Leads from the right precordium (V_{3R}, V_{4R}) are essential in the diagnosis in such instances. Displacement of the transitional zone into the left axilla also occurs; leads V_4 and V_5 are recognized as right ventricular by noting that the late intrinsicoid deflection is synchronous with that in V [illegible] and by the demonstration of predominant left ventricular patterns of opposite direction in axillary or back leads

D. QRS-T CONFIGURATION IN LEADS OVER RIGHT SIDE OF HEART

1. QRS complex. Leads just to the right of the transitional zone may show an Rs complex but those farther to the right show the classical pattern characterized by (a) an initial R wave derived from activation of the septum in a left-to-right direction (b) a late secondary upstroke (R' deflection) due to delayed activation of the free wall of the right ventricle. The intervening space between the R and R' waves may be taken up by coarse slurring or by a downstroke that may stop above the isoelectric line to form a notch or carry below to form an S wave. This downstroke reflects negative potentials transmitted from the left ventricular cavity to the right precordium in the brief interim during which activation is undergoing extinction in the septum and onset in the free wall of the right ventricle.

The upstrokes of the R and R' waves are usually smooth except for the intervening slurring or notching. Instead of a single episode of slurring or notching as described above there may be an additional notch or slur on the upstroke of the R' deflection. This second notch does not necessarily indicate a conduction defect in the free wall of the right ventricle. It may merely reflect temporary overbearance of negative potentials transmitted from the left ventricle. In this case it should be synchronous with the R peak in left ventricular leads.

The initial R wave of septal derivation is maximal in leads just to the right of the transitional zone progressively diminishes with increasing distance from the septum and may be barely detectable in leads over the right atrium and invisible in leads farther out. An apparent initial downstroke in distant leads under these circumstances is identified as an s wave not as a q wave and therefore does not carry the diagnostic implications of a q wave. Such a finding is recongized as an sR' deflection not only from the presence of rsR' complexes in leads nearer the right side of the septum but also from comparison of their durations. The time from onset of an initial upstroke to the termination of an R wave is significantly longer than the time from an initial downstroke to the termination of the QRS in leads farther to the right. This confirms disappearance of the initial septal r wave in distant leads and the identification of the recording as an sR complex. The following measurements are made in the lead where they are longest.

a. Initial upright delfection is an index of septal activation. Although the time of completion of septal activation is not definitely known in the absence of simultaneous

F QRS-T CONFIGURATION AT TRANSITIONAL ZONE When the electrode in one or more positions happens to straddle the area of precordial projection of the potential variations of the anterior end of the septum, the tracing may show a transitional QRS and/or T pattern, that may cause confusion unless interpreted in the light of the findings in adjacent right and left ventricular leads

1 QRS The usual finding in a transitional lead namely, a QRS complex of low voltage and of comparable shape to that in an adjacent right or left ventricular lead should be easily recognized Diagnostic difficulties arise when a Qr or QR pattern is recorded since the duration of the downstroke of the Q and the Q/R ratio are as great as found in association with infarction involving the entire thickness of the left ventricular wall This pattern is differentiated from that of myocardial infarction by its localization, usually to one lead and by the fact that there is an abrupt change from this very broad Q to an initial R in the next lead on the left instead of a gradual diminution of the Q wave observed as the electrode is moved away from the center of an infarct The biventricular source of the complex is established by demonstration of the correspondence of the downstroke of the Q to the descending limb of the QS in leads farther to the right and the synchrony of the late R with that in leads farther to the left

2 S-T segment and T wave The transitional zone for the T wave may lie to the right of that for the QRS as a result of a counterclockwise rotatory movement of the heart during mechanical systole, occurring between the inscription of the QRS and T waves As a consequence a lead displaying an rS, qrS or QS pattern transmitted from the anteroseptal wall of the right ventricle or a Qr pattern coming from the transitional zone may show a convex S-T segment and sharply inverted T wave similar to that in left ventricular leads or may show a fusion S-T complex characterized by an elevated S-T segment like that in right ventricular leads followed by sharp inversion of the T wave like that in left ventricular leads Unless the registration of such mixed transitional patterns in uncomplicated LBBB is appreciated an erroneous diagnosis of anterior infarction may be made

II APICAL FORM OF INCOMPLETE LEFT BUNDLE BRANCH BLOCK is characterized by (1) supraventricular origin and normal transmission of the impulse through the A-V node (2) a QRS interval of 10- 11 sec (3) an initial slurred or notched R wave in all customary leads facing the apical portion of the left ventricle (V_8 V_7 V_6 and V_5 V_4 and aV_F if left ventricular) (4) a normal q component of a qR complex in high left ventricular leads In such cases autopsy reveals left ventricular hypertrophy The septal vector is presumably directed from right to left in the apical portion to account for the initial upstroke in all customary left ventricular leads and in normal left to right direction in the basal portion to account for the initial q wave in high precordial leads The underlying incomplete left bundle branch block is confined to the apical portion of the septum and may sometimes be correlated with a lesion in this area To be differentiated is the electrocardiogram with comparable findings in the customary precordial leads but an abnormal Q component of a QR complex in high precordial leads This abnormal Q wave may be correlated with infarction of the subendocardial portion of the basal aspect of the free wall of the left ventricle as described on page 152 Infarction of the subendocardial portion of the apex usually coexists but is not manifested by Q waves in the customary precordial leads because of the right-to left vector in association with activation of the apical portion of the septum The electrocardiographic findings are comparable to those of the classical form of incomplete left bundle branch block except in high precordial leads facing the left ventricle The qR complex in these leads is 10 to 11 sec in duration and consists of a normal q wave less than 03 sec in duration and an R wave with prolonged ascending limb 04 sec or greater from onset to peak These findings are attributable to prolongation in conduction through the outer wall of the left ventricle at the base, which is usually referable to hypertrophy

primary *left ventricular hypertrophy* the R wave is tall and the intrinsicoid deflection late whereas the S wave is relatively small yet widened and slurred When incomplete RBBB is present the findings in left ventricular leads depend upon whether or not there is predominant right ventricular hypertrophy If the latter is present the r wave in left ventricular leads tends to be relatively small and brief and the S wave exaggerated if right ventricular hypertrophy is not present evidence of the right ventricular conduction defect may not be found in left ventricular leads

2 S T and T pattern In uncomplicated RBBB left ventricular leads characteristically show an isoelectric S-T junction a concave upward S-T segment and an upright T wave When there is predominant left ventricular hypertrophy the S-T segment may be depressed and convex and the T wave inverted

F QRS-T CONFIGURATION AT TRANSITIONAL ZONE In many cases of RBBB the transitional zone is broadened to extend over 2 or 3 leads Familiarity with transitional zonal patterns in uncomplicated RBBB is essential in the differentiation from coexisting anteroseptal infarction

1 QRS patterns Transitional leads may reflect predominantly the potential variations of the anteroseptal wall of (1) the right ventricle in which event an Rsr's' or a variant is recorded or (2) the left ventricle manifested by an rS deflection Towards the end of the S wave of this latter deflection there is usually notching or slurring synchronous with the R' deflection of right precordial leads The marked reduction in the amplitude of the R wave in the transition from right to left ventricular leads and the ... eft ventricle is the usual

2 S-T pattern may resemble that in leads over the right ventricle or that in leads over the left ventricle or may be a mixture of the two

VENTRICULAR HYPERTROPHY

LEFT VENTRICULAR HYPERTROPHY (LVH) is almost always but not invariably present when the electrocardiogram displays one of the following 3 patterns I Complete LBBB, II Incomplete LBBB III Prolonged conduction through the outer wall of the left ventricle manifested by (A) qR complexes in left ventricular leads consisting of a q wave less than 03 sec in duration and less than 25% of the amplitude of the succeeding upstroke and a tall unnotched R wave measuring 04 sec or more from onset to peak with a late intrinsicoid deflection beginning more than 05 sec after the onset of the QRS (B) a depressed convex S-T segment and an inverted T wave (or less typically a diphasic to flat T wave) that remain fixed in serial tracings

The foregoing 3 patterns should be considered presumptive rather than pathognomonic of LVH and require clinical support before a definite diagnosis is made because of the fact that bundle branch block may be produced by a localized lesion in the septum irrespective of the presence or absence of LVH and prolonged conduction through the outer wall may result from diffuse ischemia hyperkaliemia etc in the absence of LVH The electrocardiographic patterns associated with LVH are independent of the etiology (i e , the patterns of LVH due to hypertension rheumatic aortic valvulitis and syphilitic aortic insufficiency are indistinguishable) and may occur in the absence of anatomically demonstrable lesions in the left ventricle other than hypertrophy

The pattern designated as prolonged cond ... must be differ... outer wall o... infarction) ... lesion (usually ...ested by a preliminary downstroke a secondary upstroke and a late intrinsicoid deflection but is distinguished either by a Q wave that is 03 sec or more in duration and more than 25% of the amplitude of the R wave or by a qR complex with one

leads from the right ventricular cavity it is recommended that the measurement be made to the onset of the R' wave in the presence of a notched or slurred upright QRS If there is an S wave intervening between the R and R' wave the time from the onset of the R to the point where the string crosses the isoelectric line in the course of the downstroke of the S wave is recommended as an index of septal activation This measurement should amount to 04 sec or longer in complete right bundle branch block and is usually between 02 sec and 04 sec in incomplete right bundle branch block

b <u>Late peak of the R' wave</u> The time from onset of the QRS to the peak of the R' (i e onset of the intrinsicoid downstroke) is an index of the time required for the activation of the septum plus the free wall of the right ventricle This measurement should amount to 08 sec or more with complete RBBB, 06 to 08 sec with incomplete RBBB

The R' deflection tends to attain its maximum in leads near the tricuspid ring or crista supraventricularis and progressively diminishes as the electrode is moved to right or left With complete RBBB, the time interval from onset to peak of the maximal R' is generally 04 sec or more due to aberrant right ventricular activation irrespective of whether or not right ventricular hypertrophy or damage are anatomically demonstrable Furthermore, the R' deflection may be of high voltage even in the absence of right ventricular hypertrophy because of the fact that activation is still in progress in the free wall of the right ventricle after the left ventricle has become depolarized and no longer creates opposing forces With incomplete RBBB however the duration and amplitude of the ascending limb of the maximal R' serve as an index of the physiologic state of the free wall of the right ventricle A time interval from onset of R' to its peak, amounting to 03 sec or longer and R' amplitude exceeding 10 mm are usually referable to right ventricular hypertrophy but are occasionally due merely to delayed conduction in the absence of hypertrophy When incomplete right bundle branch block is the result of temporary prolongation in conduction through the septum and right ventricle, as in some cases of acute cor pulmonale, the transient basis for the lesion is established by its disappearance in serial tracings

2 <u>S-T segment and T wave</u> are characteristically discordant with the QRS but are occasionally concordant for no demonstrable anatomic reason The typical findings in right ventricular leads are depression of the S-T junction upward convexity of the S-T segment and deep inversion of the T wave

E QRS-T CONFIGURATION IN LEFT VENTRICULAR LEADS

1 <u>QRS</u>

a <u>Direction of the initial deflection</u> Septal activation exclusively in a left-to-right direction might be expected to exaggerate the normal q wave recorded in left ventricular leads and together with late activation of the free wall of the right ventricle tends to reciprocally decrease the amplitude of the R wave in the same leads Thus Q/R ratios exceeding 25% are observed in left ventricular leads of patients with RBBB in the absence of infarction however the time interval from onset to nadir of the q wave remains within normal limits of 02 sec On the other hand an initial upstroke may be recorded in leads over the anterior wall of the left ventricle and only a minute q in other left ventricular leads presumably due to unusually early onset of activation of the free wall of the left ventricle

b <u>R/S relationships</u> In the presence of complete RBBB leads over the left ventricle characteristically show a relatively narrow R and broad S When primary right ventricular hypertrophy is responsible for the RBBB the ascending limb of the r wave in left ventricular leads is unusually small in amplitude and brief in duration whereas the S is deep as well as broad When the RBBB is a complication of a

normal perhaps because of the lengthening of the Purkinje network secondary to left ventricular enlargement Nevertheless the q wave of uncomplicated LVH should conform to the following criteria that differentiate it from the abnormal Q wave associated with subendocardial infarction

1) *Time from onset to nadir — less than 03 sec*

2) Relative amplitude —less than 25% of succeeding R

b Characteristics of the R wave in leads with a qR pattern The most important feature is lengthening in the interval between onset and peak of R to 04 sec or more This is often accompanied by slurring of the ascending limb of the R wave on the other hand distinct notching of the upstroke of a qR complex requires classification into another category mentioned above and discussed later The prolongation in the ascending limb of the R wave is presumably a manifestation of the increased thickness of the left ventricle and is largely responsible for another characteristic feature — the late intrinsicoid deflection, beginning more than 05 sec after the onset of the QRS The average amplitude of the left ventricular R wave is greater in LVH than in normals reflecting a greater voltage developed during activation of a thicker wall, but considerable overlapping occurs among individual cases Amplitudes exceeding 26 mm in V_5 have been found in approximately 20% of patients with established LVH but are rare in normals This observation is more suggestive of the need for careful temporal measurements than of primary diagnostic import most high voltage R waves due to LVH show prolongation of the duration of the ascending limb

2 S T segment and T wave The characteristic findings are depression of the S-T junction, upward convexity of the S-T segment and inversion of the T wave The findings are subject to considerable variation as follows

a S-T junction and segment show slight to moderate depression They are rarely isoelectric except in early or complicated cases and are not elevated unless there is a superimposed lesion such as acute subepicardial injury Marked depression of the junction together with straightening of the segment may result from digitalization in which event the Q-T interval is short or from acute subendocardial injury, in which the Q-T interval is long and the pattern subject to rapid serial changes The S-T segment shows convex bowing in uncomplicated LVH with inversion of the T wave but is generally concave when the T wave is upright

b T wave

1) Variations in direction and contour in single tracings Although inversion is the typical finding the T waves may be diphasic, flattened or low upright The latter do not preclude the diagnosis of LVH when the classical QRS changes are present but forestall even a suspicion of the diagnosis if the classical QRS changes are absent The T waves may be as sharply inverted and as deep as those commonly found during organization of myocardial infarction or during the subacute phase of pericarditis but are distinguished by the fact that the S-T junction and segment are usually depressed in uncomplicated LVH but are elevated or at least isoelectric in subepicardial injury

The T waves are usually more inverted in leads from the axilla or back with a qR pattern than in leads from the anteroseptal wall with an Rs pattern but a progressive decrease in the depth of the inverted T wa- [illegible]

or more definite notches on its ascending limb The notching of the upstroke of an R wave that is part of a qR complex an abnormal finding should not be confused with notching of the upstroke of an R wave that is part of an Rs complex which may be a normal variant in leads near the transitional zone This pattern will be discussed in more detail in the section on myocardial *infarction*

On the other hand many patients with uncomplicated LVH fail to show any of the 3 electrocardiographic patterns cited above as presumptive of LVH In some of the latter, the electrocardiogram is within normal limits, but in a relatively large group it shows IV a borderline pattern characterized by S-T and T abnormalities like those of group III but without QRS changes indicative of prolonged activation Pattern IV, as an isolated finding may be regarded as suggestive of LVH, but requires definite supportive clinical evidence to become presumptive of LVH The characteristics of these 4 patterns are as follows

I COMPLETE LBBB – pages 125-128

II INCOMPLETE LBBB – pages 125-128

III PROLONGED CONDUCTION THROUGH OUTER WALL OF LEFT VENTRICLE

A QRS DURATION —usually 10 to 12 sec , but may be slightly longer than 12 sec or less than 10 sec

B Q-T DURATION – typically beyond the upper limits of normal

C LOCATION OF TRANSITIONAL ZONE – may be in the usual normal position between V_2 and V_4 but is often displaced to the left between V_4 and V_5 not uncommonly to the axilla between V_5 and V_6 and occasionally as far as V_7

D QRS-T CONFIGURATION IN LEADS OVER THE RIGHT SIDE OF THE HEART

1 QRS *is roughly reciprocal to that in leads over the left ventricle The usual finding* is a small r an early intrinsicoid deflection and an abnormally deep and prolonged S wave the time from onset of the QRS to nadir of the S wave exceeding 05 sec In leads over or beyond the right atrium a QS deflection may be recorded as a variant presumably from fading of positive potentials transmitted from the right side of the septum and epicardial surface of the right ventricle and improved transmission of negative left ventricular cavity potentials through the mitral orifice and left atrium and thence to the right atrium The positive potentials may give rise to slurring or notching near the base of the descending limb of the QS In the presence of such QS complexes consideration must be given to the possibility of a right-to-left septal vector indicated by the registration of an initial upstroke rather than a q wave in every left ventricular lead

2 S-T complex The junction is elevated often more than the normal limit, the segment is usually curved with upward concavity and the T wave is upright and tall

E QRS-T CONFIGURATION IN LEFT VENTRICULAR LEADS

1 QRS is manifested by an Rs or qRs pattern in leads over the anteroseptal wall and by a qR pattern in leads over the lateral and posterior walls Measurements of duration of individual phases should be made in the lead with a qR pattern in which the time intervals appear greatest

a Direction of the initial deflection A q wave is often absent from leads over the anteroseptal wall of the left ventricle but should always be demonstrable in leads over the lateral and/or posterior wall The amplitude of this q wave may be exaggerated beyond the usual normal limit of 3 mm because of the greater voltage developed during activation of the hypertrophied septum and improved transmission to the axilla through closer approach of the enlarged left ventricle to the thoracic cage Furthermore the duration of this q wave may be slightly greater than

normal, perhaps because of the lengthening of the Purkinje network secondary to left ventricular enlargement Nevertheless the q wave of uncomplicated LVH should conform to the following criteria that differentiate it from the abnormal Q wave associated with subendocardial infarction

1) Time from onset to nadir ~less than 03 sec

2) Relative amplitude ~less than 25% of succeeding R

b Characteristics of the R wave in leads with a qR pattern The most important feature is lengthening in the interval between onset and peak of R to 04 sec *or more* This is often accompanied by slurring of the ascending limb of the R wave, *on the other* hand distinct notching of the upstroke of a qR complex requires *classification into* another category mentioned above and discussed later The prolongation in the ascending limb of the R wave is presumably a manifestation of the increased thickness *of the left* ventricle and is *largely responsible* for another characteristic feature ~ *the late intrinsicoid* deflection beginning more than 05 sec after the onset of the *QRS The* average amplitude of the left ventricular R wave is *greater* in LVH than in *normals*, reflecting a greater voltage developed during *activation* of a thicker wall, but *considerable* overlapping occurs among individual cases *Amplitudes exceeding* 26 *mm in* V_5 *have* been found in approximately 20% of patients with established LVH but are rare in normals This observation is more *suggestive of the need for careful temporal* measurements than of *primary diagnostic import most* high voltage R waves due to LVH show prolongation of the duration of the *ascending limb*

2 S T segment and T wave The characteristic findings are depression of the S-T junction, upward convexity of the S-T segment and *inversion* of the T wave The findings are subject to considerable variation as follows

a *S T junction* and segment show *slight to moderate depression* They are rarely isoelectric except in early or *complicated cases, and* are not elevated unless there is a superimposed lesion, such as *acute subepicardial injury* Marked depression of the junction together *with straightening of the segment* may result from digitalization, in which event the Q-T interval *is short or from* acute subendocardial injury, *in which* the Q-T interval is long and the *pattern subject* to rapid *serial* changes *The S-T* segment shows convex *bowing in uncomplicated LVH with* inversion of the T wave, but is generally concave when the T wave is upright

b T wave

1) [illegible]

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[illegible] of LVH when the *classical QRS* changes are present, but forestall even a *suspicion* of the diagnosis if the *classical QRS* changes are absent The T waves *may* be as sharply inverted and as deep as those commonly found during organization of myocardial *infarction or during* the subacute phase of pericarditis but are distinguished by the fact that *the S-T junction* and segment are usually depressed in uncomplicated LVH but are *elevated, or* at least isoelectric in subepicardial injury

The T waves are usually more inverted in leads *from the* axilla or back with a qR pattern than in leads from the anteroseptal wall with an Rs pattern, but a progressive decrease in the depth of the inverted T waves, along *with* a decrease in QRS voltage occurring as the electrode is moved leftward from *the* V_4 position *is* an occasional variant attributable to loss of potential with increasing distance *from* the heart

or more definite notches on its ascending limb The notching of the upstroke of an R wave that is part of a qR complex an abnormal finding should not be confused with notching of the upstroke of an R wave that is part of an Rs complex which may be a normal variant in leads near the transitional zone This pattern will be discussed in more detail in the section on myocardial infarction

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RIGHT VENTRICULAR HYPERTROPHY (RVH) may be accompanied by one of the following patterns I Complete RBBB II Incomplete RBBB III Prolonged conduction through the tricuspid ring and/or crista supraventricularis, IV Pattern suggesting right ventricular dilatation but not hypertrophy V Tracings showing no evidence of a right ventricular lesion The significance of each pattern will be brought out individually The electrocardiographic patterns associated with RVH are independent of etiology (i e the patterns of pulmonary stenosis mitral stenosis cor pulmonale may be indistinguishable) and may occur in the absence of anatomically demonstrable lesions in the right ventricle other than hypertrophy

I COMPLETE RBBB is encountered more often as a complication of failure from primary left ventricular disease than as a manifestation of isolated right ventricular hypertrophy or damage The most helpful evidence in the differentiation of the 2 major causes of uncomplicated RBBB is obtained from the QRS pattern in left ventricular leads A qRs complex in these leads with prolonged upstroke late intrinsicoid deflection and relatively small s indicates underlying LVH whereas a small r (or qr) early intrinsicoid deflection and deep broad S suggest primary RVH as the cause of the RBBB

II INCOMPLETE RBBB Attention should be directed towards the duration and amplitude of the ascending limb of the R' deflection It is important that sufficient leads be taken to cover the tricuspid ring and crista supraventricularis in order to find the lead with the largest R' and absence of S' This lead is used for measurements Although the largest R' is usually recorded in V_1 or HV_2, it may be found in V_{3R} or V_{4R} thus emphasizing the need of right precordial leads A time interval from onset of R' to peak of R' amounting to 03 sec or longer along with increased amplitude of R' (10 mm or more) and diminution or absence of s' is usually referable to RVH but may be a manifestation of prolonged conduction through the outer wall of the right ventricle in the absence of hypertrophy

III PROLONGED CONDUCTION THROUGH THE OUTER WALL OF THE RIGHT VENTRICLE in the absence of RBBB is usually but not invariably due to RVH It is characterized by a distinctive electrocardiographic pattern that may be considered at least presumptive of RVH

A QRS duration – 08 to 11 sec

B Q T interval – characteristically near or above upper limits of normal

C LOCATION OF TRANSITIONAL ZONE The most striking change in pattern usually does not occur at the interventricular septum but rather at the boundary between the tricuspid ring or crista supraventricularis on the one hand and the trabecular portion of the right ventricle on the other The frontal projection of this boundary in the fourth and fifth interspaces is almost invariably to the right of V_3 usually to the right of V_2 and not uncommonly to the right of V_1 The boundary line tends to extend diagonally upward and towards the left so that in the third interspace it is usually between the HV_2 and [illegible] positions but sometimes between HV_1 and HV

Right ven- [illegible]
ring and/or [illegible]
source of t[illegible] structures is the apparent [illegible] that constitutes the essential feature of the rsR' rsR's qR or qRs complex recorded in leads to the right of this boundary line in typical cases of RVH

The trabecular portion (i e the body and apex) of the right ventricle remains relatively thin in most cases of RVH and as a consequence overlying leads usually display an rS complex that contrasts strikingly with the pattern in leads farther to the right The transitional zone between the trabecular portion of the right ventricle and the anteroseptal wall of the left ventricle is gradual and often difficult to localize since rS complexes constitute the usual major feature of the findings over both structures The variations in pattern encountered at this transitional zone will be discussed below

The inverted T waves so commonly associated with left ventricular hypertrophy are indicative of reversal in the direction in repolarization which, in turn, is presumably secondary to prolongation in activation Deeply inverted T waves have been recorded in left ventricular leads in cases in which subsequent autopsy revealed no coronary narrowing or myocardial lesions From the findings in a single tracing this pattern should not be attributed to ischemia or "left ventricular strain " The latter term is objectionable because of a physiologic connotation beyond the realm of electrocardiography

2) Variations in serial electrocardiograms Tracings taken at long intervals during the development of an otherwise uncomplicated left ventricular hypertrophy tend to show a very gradually increasing S-T depression accompanied by a change from a low upright T to a flattened diphasic and then inverted T Abnormalities in S-T pattern usually precede those in the QRS Tracings taken at short intervals during exercise anoxia, or during a clinical episode of left ventricular failure may show the following significant transient changes in S-T and/or T wave without changes in the QRS complex

a) Transient S-T depression of 5 mm or more, together with a horizontal or downwardly sagging course of the S-T segment, is indicative of acute subendocardial injury from ischemia and will be discussed at more length later Abnormal S-T displacement is frequently, but not necessarily, accompanied by increasing negativity of the T wave

b) Transient change from an inverted to upright T wave, accompanied by Q-T prolongation but not by alteration in the S-T junction, may be referable to a lesser degree of subendocardial injury or may be reciprocal to an acute lesion of the opposite wall

c) Transient inversion or increase in depth of an inverted T wave, accompanied by Q-T prolongation, but not by alteration in the S-T junction may be referable to increased delay in repolarization of the subepicardial layer Although these changes are frequently designated as "acute left ventricular strain " the term is not appropriate because of its mechanical implication

F QRS-T CONFIGURATION IN TRANSITIONAL LEADS

1 QRS The usual finding at the transitional zone is a QRS complex of relatively low voltage consisting of R and S deflections that are reduced in amplitude as contrasted with the QRS complexes in adjacent leads to the right and left These intermediate complexes often exhibit notching or coarse slurring which can be recognized as a transitional zonal phenomenon by demonstration of synchrony with an R peak in a right or left ventricular lead Occasionally right ventricular activation is sufficiently ahead of left so that leads at the transitional zone yield a quadriphasic rsR's' complex

2 S-T Since the transitional zone for the T may be located to the right of that for the QRS leads with right ventricular or transitional QRS patterns may have T waves that resemble those in left ventricular leads or display fusion patterns as described in connection with LBBB page 69

IV BORDERLINE PATTERNS In this group are included tracings with S-T and T patterns like those of group III but without prolongation in the duration of the ascending limb of the R wave or delay in onset of the intrinsicoid deflection

V NORMAL PATTERNS may be found in association with left ventricular hypertrophy demonstrated by clinical or post-mortem examination

2) Characteristics of the late R (or R') The time from onset to peak of this late R (or R') is increased to 03 sec or greater and is abnormally long for passage of the impulse from endocardial to epicardial surface of the muscular tricuspid ring or crista supraventricularis This is usually due to hypertrophy but occasionally results from a lesion prolonging conduction in the absence of hypertrophy (e g as in acute cor pulmonale) The time from the beginning of the QRS to the onset of the intrinsicoid downstroke is also increased to 04 sec or longer The lateness of the intrinsicoid downstroke is also shown by the fact that it either terminates at the isoelectric line or is followed by an abnormally small s (or s') wave A terminal s (or s') deflection is generally recorded in leads near the midline of the body with a late R and disappears as the electrode is moved towards the right

b S T segment and T wave The characteristic findings are depression of the S-T junction upward convexity of the S-T segment and inversion of the T wave, but a number of variants occur The S-T junction may be isoelectric or even slightly elevated these findings along with a convex S-T segment and a sharply inverted T wave arouse the suspicion of complicating subepicardial injury but may occur in the absence of an anatomically demonstrable lesion other than hypertrophy The T wave may be diphasic flattened or occasionally upright in which event it is accompanied by a concave S T segment

2 Patterns recorded over the trabecular portion of the right ventricle to the left of the tricuspid ring and to the right of the septum The P waves in these leads are upright with gently sloping limbs

a QRS patterns may resemble those in leads from the tricuspid ring and/or crista supraventricularis when the electromotive forces developed during activation of these structures are sufficiently great to carry to the left of their usual precordial projections or when there is an accompanying marked hypertrophy of the body of the ventricle However the usual finding is a sharply contrasting pattern consisting of either an rS an rSr' a QS qrS or qrSr' complex

The direction of the initial deflection depends upon the direction of the septal vector When this extends from left to right an initial comparatively small r wave is inscribed partly from septal activation partly from passage of the impulse through the relatively thin trabecular portion of the right ventricle The intrinsicoid deflection is early in onset and is followed by a deep S with or without a terminal small r' presumably transmitted from the epicardial surface of the tricuspid ring or crista supraventricularis

When the septal vector passes from right to left the direction of the initial deflection depends upon whether the negative right ventricular cavity potentials or the positive potentials reaching the surface from early activation of the free wall have the preponderant influence on the tracing In the latter event an rS (or rSr') complex is recorded in the former a QS or qrSr' complex is registered On the downstroke of these QS complexes there is usually a notch or coarse slurring corresponding to an embryonic r wave produced by activation of the thin trabecular wall of the right ventricle a late notch or slurring near the end of the upstroke of the QS synchronous with the R in leads further to the right comes from late activation of the tricuspid ring or crista supraventricularis

These QS or qrS deflections resemble those in left ventricular leads taken over areas of transmural infarction the illusion is made even more realistic by the fact that the S T segments are usually isoelectric or elevated and the T waves are often sharply inverted The problem is made even more difficult by the fact that the pattern recorded in different leads over the trabecular portion of the right ventricle may vary from qrS to QS to rS depending upon distance from septum and the basal

D QRS-T CONFIGURATION IN LEADS OVER THE RIGHT SIDE OF THE HEART As indicated above, a sharp contrast is likely between patterns recorded over or to the right of atrioventricular groove on the one hand and patterns recorded over the body of the right ventricle on the other requiring separate description

1 Patterns recorded over or to the right of the tricuspid ring and crista supraventricularis (Leads V_{4R}, V_{3R}, generally V_1 HV_1 HV_2) The diphasic contour of the P wave aids in determining the anatomic relations of the electrode

a QRS

1) Direction of initial deflection — may be upright or inverted depending upon direction of mean vector associated with septal activation and upon distance between electrode and septum Since the potentials derived from septal activation and transmitted to the right precordium progressively diminish with increasing distance, the direction of the initial deflection should be determined in the lead nearest the septum that displays a late R wave

a) Septal vector directed from left to right causes initial positivity of the right ventricular cavity (as indicated by the registration of a small initial r wave through leads taken with an electrode in the cavity) and should therefore produce an initial upstroke, at least in the nearest lead with a late R' wave The initial r of septal origin is small and brief and is separated from the final R' by slurring or by a downstroke that usually stops above the isoelectric line to form a notch that may extend slightly below to form a minute s wave This results in either an rR' complex (with or without an s') or an rsR' pattern (with or without an s')

The general contour resembles that of incomplete RBBB The difference lies in the duration of the initial r wave, as measured from the onset of the QRS to the onset of the R' in the presence of rR' patterns or to the point where the string crosses the isoelectric line in the downstroke of the s component of an rsR' complex This interval is between 03 and 04 sec in incomplete RBBB and is 02 sec or less in the pattern now under discussion As the electrode is moved to the right the initial r decreases and may disappear in Lead V_{4R} or in leads farther to the right to leave a small initial downstroke and a large late upstroke which should be identified as an sR' complex rather than as a qR complex

b) Septal vector directed from right to left occurs in some cases of preponderant RVH (in the absence of LBBB) as shown by the fact that all records from the right ventricular cavity in these cases display a QS pattern, whereas all right precordial leads with a late R wave display either a qR a qRs or a QR pattern As indicated by the use of either lower and upper case letters to characterize the initial deflection it is subject to considerable variation both in amplitude and duration In some cases the time from onset to nadir of Q is sufficiently long and the Q/R ratio sufficiently great to mimic the abnormal QR pattern recorded in left ventricular leads over an area of subendocardial infarction

The qR or QR pattern recorded in leads near the right atrioventricular groove as a result of the combination of right to left septal activation with hypertrophy of the tricuspid ring occurs in the absence of myocardial infarction but must be differentiated from the qR or QR pattern recorded in the same leads as a result of septal infarction with RBBB In the former the QRS interval is almost always less than 12, in the latter it is 12 sec or longer The S-T patterns are of aid provided that the infarct is acute Furthermore septal infarction usually continues into the anterior wall of the left ventricle to produce diagnostic patterns in leads to the left of the transitional zone (page 150)

right ventricular hypertrophy This may result in qr ratios well above 25 However a mistaken diagnosis of anteroseptal infarction should be avoided by noting that the time from onset to nadir of q is normal (i e 02 sec or less) and by noting other signs of the causative right ventricular hypertrophy namely a prominent S in left ventricular leads the counterpart of the diagnostic late R of leads from the base of the right ventricle

b Relationships of R to S waves depend upon the relative magnitude and timing of the forces developed in activation of the right and left ventricles With right ventricular hypertrophy and a normal left ventricle rS complexes may be recorded in all left ventricular leads or the r wave may progressively increase at the expense of the S, as the electrode is moved into the axilla and left back to result in a gradation from rS to Rs to perhaps qRs complexes The onset of the intrinsicoid deflection in left ventricular leads is significantly earlier than in those from the base of the right ventricle With a preponderant RVH and slight LVH the findings in left ventricular leads may be like those of the second normal pattern described above or rarely, may be indicative of coexistent LVH

2 S T and T wave The typical findings are isoelectric to slightly elevated S-T junction concave segment and upright T wave With associated LVH S-T depression and T wave inversion may be observed

F QRS T CONFIGURATION IN TRANSITIONAL LEADS IN THE VICINITY OF THE INTER-VENTRICULAR SEPTUM The shift from predominant right to left ventricular leads is often gradual with no clearly defined transitional complexes The presence of one or more transitional complexes may lead to an erroneous diagnosis of anteroseptal myocardial infarction because of the tendency to diminution or extinction of the initial r wave at the transitional zone and the tendency towards registration of fusion S T and T patterns

1 QRS

a Localized reduction in the amplitude of the initial r wave or replacement [illegible] QS be observed at the transitional zone in right [illegible] due to the tendency towards mutual extinctio[illegible] vation of the septum from its right and left sides The rS or QS is recognized as a transitional zonal phenomenon by its relatively low voltage in comparison with adjacent leads to the right and left *Patterns of this type are subject to fluctuation with respiratory shifts in cardiac position and may show phasic variations from rS to QS to Rs complexes as rotation on a longitudinal and/or anteroposterior axis with descent and ascent of the diaphragm brings successively the right ventricle transitional zone and left ventricle under the electrode Further aid in the differentiation of these patterns from the QS due to transmural infarction is furnished by the QRS configuration in leads to the left of that in which the QS is recorded* If a transmural infarction were responsible for the QS patterns one would expect the infarct to continue subendocardially for a sufficient distance to produce abnormal QR patterns in one or more leads to the left

b Fusion QR pattern is a rare variant that may be inscribed in a single lead when the electrode happens to straddle the septum in a patient where QS patterns are recorded in leads over the adjacent right ventricular wall and R or Rs patterns over the adjacent left ventricular wall This QR pattern is recognized as a transitional zone phenomenon by (1) its relatively low voltage in comparison with adjacent leads to right and left (2) the tendency to fluctuation with respiratory shifts in cardiac position (as discussed above) and (3) its strict localization as shown by normal left ventricular patterns in leads to the left To verify the latter it is advisable to take additional leads in the immediate vicinity of that in which the QR pattern is recorded

muscular ring Nevertheless, these patterns may be differentiated from those of infarction of the anteroseptal wall of the right ventricle by the following criteria (a) the demonstration of evidence of RVH in leads over the tricuspid ring should make one hesitate to ascribe QS patterns in nearby leads to infarction (b) the identification of the electrode positions from which the QS or qrS patterns are recorded as right rather than left ventricular, (c) the absence of signs of subendocardial destruction in leads over the anteroseptal wall of the left ventricle If a transmural infarct were responsible for QS patterns, one would expect the infarct to continue subendocardially for a sufficient distance to produce abnormal QR patterns in leads to the left of those showing QS patterns

b S-T and T waves are subject to considerable variation, not only in different cases, but also in the same patient during and after recovery from an acute right ventricular load caused by pulmonary embolism extensive pneumonia, intercurrent left-sided failure etc The S-T junction may be isoelectric to depressed and followed by a convex segment and inverted T, as in leads over the tricuspid ring or crista supraventricularis On the other hand, the S-T junction may be isoelectric to elevated and accompanied by a concave segment and upright T wave as in the normal or by a convex segment and sharply inverted T wave as in leads over the base of the right ventricle The latter S-T and T pattern is similar in contour to that associated with recent anteroseptal infarction but differs in distribution

In primary right ventricular lesions, the maximal S-T elevation and most striking cove inversion of the T wave occurs in leads over the tricuspid ring and gives way to isoelectric S-T segments and upright or shallowly inverted T waves in leads to the left of the septum, in recent anteroseptal infarction S-T elevation and cove inversion of the T waves may be found in leads over the right ventricle presumably from the septal portion of the infarct, but is almost always more striking in leads just to the left of the transitional zone over the anteroseptal wall of the left ventricle Shortly after an acute right ventricular load as from pulmonary embolism elevated convex S-T segments and sharply inverted T waves are likely to be found in right ventricular leads, followed by rapid return to upright T waves and concave segments in serial tracings S-T depression in leads from the posterolateral wall may accompany the elevation in right ventricular leads and tends to return to the isoelectric line concurrently Although such S-T depression might be reciprocal to the elevation, it is often too great and too extensive to be explained on this basis In acute cor pulmonale there is evidence of interference with circulation through the right coronary artery secondary to the right ventricular dilatation, this provides a basis for the interpretation of undue S-T depression in leads from the posterior and lateral walls as an expression of acute subendocardial injury from ischemia The S-T evolution in acute cor pulmonale is more rapid than that in anteroseptal infarction The differential diagnosis is also facilitated by the differences in QRS pattern and in the distribution of the sharply inverted T waves described above

E QRS-T CONFIGURATION IN LEFT VENTRICULAR LEADS

1 QRS The patterns depend upon the direction of the septal vector and the relative magnitude of the forces developed in activation of the right and left ventricles

a Direction of initial deflection depends upon the direction of the septal vector and the earliness of the arrival of the impulse in the free wall of the left ventricle If the septal vector passes from right to left, an initial upstroke will be expected in left ventricular leads If the septal vector points from left to right a q wave will be expected in left ventricular leads but may be obliterated by the tendency towards early arrival of the impulse in the free wall of the left ventricle If a q wave is recorded diagnostic difficulties may arise because of the tendency toward reciprocal decrease of the amplitude of the R wave in left ventricular leads in the presence of

2. Q T interval is characteristically prolonged above the upper limits of normal due to delay in repolarization of the subepicardial layers

3. S T junction S T segment and T wave. The electrocardiographic diagnosis is based on the characteristic nature of the changes in the ST-T complex and their rapid evolution in serial tracings. For convenience of description the sequential changes are arbitrarily divided into the followed three stages

a. Acute stage. The characteristic features of the acute stage are an elevation of the S-T junction an upward displacement of the S-T segment, and a monophasic upright T wave. The degree of elevation of the S-T segments and T waves is related to the degree of injury. The S-T segment generally ascends in a straight line or sometimes in a curved line with upward concavity and less commonly exhibits the upward convexity characteristic of the S-T segment in myocardial infarction. Early in the evolution the T waves may be tall upright with sharp peaks and narrow bases. Such T waves bear a resemblance to those of hyperkaliemia and may be an expression of escape of potassium out of the cells into the interstitial spaces. these T waves are differentiated from those due to potassium intoxication by the characteristic S-T segment elevation in the former, but not in the latter. Soon in the course of evolution the summit of the T waves becomes rounded and the terminal portion may assume a rippled contour typical of pericarditis

In the classical case, the foregoing changes in the S-T segment and T wave are present in all leads facing the injured epicardial surface. When abnormal elevation of the S-T junction occurs in only a few of the precordial leads as a result of pericarditis it is rare to find reciprocal depression of the S-T junction in the remaining leads. Absence of reciprocal depression aids in differentiating S-T elevation due to pericarditis from S T elevation due to myocardial infarction.

The S T elevation of acute pericarditis is usually quite transient, lasting from a few hours to a few days. In serial electrocardiograms taken during this period, progressive return of the S-T junction back to the isoelectric line may be demonstrated. With transitory types of inflammatory or traumatic pericarditis the T wave may remain upright after the S-T junction becomes isoelectric and may fail to show the evolutionary changes described below under the subacute phase

b. Subacute stage. The transition from acute to subacute pericarditis is gradual. For descriptive purposes the return of the S-T junction to the isoelectric level is arbitrarily taken as the dividing line between the acute and the subacute stage. As the pattern passes from the acute to the subacute stage the S-T junction approaches the isoelectric line the T wave flattens and then becomes inverted. Inversion of the T wave tends to occur in all leads and is generally greatest in leads which previously had shown the greatest S T displacement. Progressive deepening of the inverted T is observed in serial tracings to reach a maximum following which there is gradual regression. The characteristic evolution may be interrupted by recurrence of the acute stage of S-T elevation presumably associated with exacerbation in injury to the subepicardial myocardium. The subacute phase ends either with the return of the S-T segment and T wave to normal or with the development of a fixed abnormality in ST-T which fails to undergo further change in serial electrocardiograms

In many cases of subacute pericarditis the T wave closely resembles the cove-plane T wave of myocardial infarction in that the S-T segment is dome-shaped with upward convexity and is followed by a sharply inverted T wave with steep concave descending and ascending limbs. The points which aid in distinguishing between subacute pericarditis and myocardial infarction are

1) QRS complexes. In myocardial infarction, the QRS is almost always abnormal either in contour or duration whereas in pericarditis the QRS is generally normal in these respects. Infarction of the free wall of the left ventricle with maintenance

2 S-T complex and T wave The main difficulty in the interpretation of transitional zone effects arises in atypical cases when leads well to the right of the transitional zone show elevated S-T segments and upright T waves and those to the left show inverted T waves The counterclockwise rotation accompanying mechanical systole (that takes place between the registration of QRS and T waves) may shift the heart sufficiently in respect to the anterior chest wall so that the left ventricle comes beneath electrode positions that faced *the septum or adjoining wall of the right ventricle* during registration of the QRS The suspicion of anteroseptal infarction may be aroused by the combination of QS complexes of right ventricular or transitional origin with inverted T waves transmitted from the left ventricle or fusion S-T complexes composed of an elevated S-T junction of right ventricular origin with inversion of the terminal portion of the T of left ventricular origin The differentiation of this transitional pattern from anteroseptal infarction is based largely on the findings in leads just to the left of that in which the QS complex is recorded The absence of an abnormal QR pattern is strongly against anteroseptal infarction

IV PATTERNS SUGGESTING RIGHT VENTRICULAR DILATATION BUT NOT HYPERTROPHY Acute right ventricular dilatation associated with acute cor pulmonale may be complicated by transient RBBB *and/or transient prolongation in conduction through the outer wall of the right* ventricle, in which event the electrocardiographic findings may transiently conform with groups I, II, or III, described above In the absence of an intraventricular conduction defect right ventricular dilatation may be manifested by QRS-T patterns over the anterior aspect of the chest, resembling those recorded over the trabecular area of the right ventricle, described above (group III), and also by transitional zone patterns like those described above For the electrocardiographic features and differentiation from anteroseptal infarction reference should be made to the foregoing description

V TRACINGS SHOWING NO EVIDENCE OF A RIGHT VENTRICULAR LESION may be encountered particularly when right ventricular hypertrophy is secondary to failure complicating primary and predominant left ventricular hypertrophy and sometimes when right ventricular hypertrophy and dilatation represent the primary and major lesion Thus, lack of electrocardiographic evidence does not exclude even primary right ventricular hypertrophy

MYOCARDIAL INJURY LOCALIZED TO THE SUBEPICARDIAL OR SUBENDOCARDIAL LAYERS

I SUBEPICARDIAL INJURY

A SUBEPICARDIAL MYOCARDITIS is manifested by an electrocardiographic pattern which is customarily referred to as the pattern of pericarditis, but is actually an expression of a lesion of the superficial layers of the myocardium This is borne out by the presence of the pattern in cases where autopsy showed subepicardial myocarditis without associated pericarditis and by its absence in cases with lesions limited to the pericardium The characteristic pattern may occur not only with inflammatory invasion of the subepicardial myocardium but also with uremic pericarditis, hemopericardium neoplastic infiltration of the subepicardium and *subepicardial infarction When the subepicardial layer is injured* in the process of pericarditis both ventricles are involved and the electrocardiographic abnormalities are found in leads over the right as well as the left ventricle In primary subepicardial myocarditis, the left ventricle is *often* involved to a greater extent than the right and the electrocardiographic abnormalities may be limited to leads over the left ventricle Although the electrocardiographic changes are actually referable to injury of the superficial layer of myocardium the customary terminology of pericarditis will be used for the pattern for purposes of convenience

1 QRS complex may decrease in voltage but shows *no abnormality in contour or duration* except in the presence of an antecedent or coexistent lesion of the deeper layers of myocardium If former curves are available for comparison the superimposed pericarditis is more readily diagnosed

B SUBEPICARDIAL INFARCTION

1 QRS complex over the area of subepicardial infarction commonly shows a reduction in amplitude of the R wave when compared with a tracing taken prior to infarction Unlike pericarditis an infarct that is limited to the subepicardial layer in one portion of the heart frequently extends into deeper layers in adjacent areas giving rise to an initial q wave followed by a notched or coarsely slurred R wave, characteristic of mid-zonal involvement or a QR pattern typical of subendocardial involvement in leads from the adjoining area

2 Q-T interval is characteristically above the upper limits of normal

3 S T junction, S T segment and T wave changes in leads over the area of subepicardial infarction are similar to those described above for pericarditis

II ACUTE SUBENDOCARDIAL INJURY is usually the result of acute ischemia and is associated with spontaneous angina The tendency for localization in the subendocardial portion is in part attributable to the poorer blood supply of this layer, in part referable to the greater compression to which the vessels in this layer are subjected during systole The cardinal electrocardiographic manifestation is an acute transitory horizontal or downwardly sagging depression of the S-T segment of 5 mm or more usually accompanied by lengthening of the Q T interval and sometimes by change in the direction of the T wave

Subendocardial ischemia may be classified according to electrocardiographic findings and his ologic distribution as patchy or diffuse In the patchy variety minute foci of ischemic muscle are interspersed between bands of intact muscle as a consequence of the latter the response to the activating impulse is not significantly altered and the QRS complex undergoes no changes In diffuse subendocardial injury the subendocardial layer fails to respond to the activating impulse and an abnormality in the QRS develops If septal activation does not change from the usual left to right direction the Q wave in left ventricular leads overlying the ischemic area becomes abnormally long in duration and large in amplitude in proportion to the succeeding R wave If septal activation is reversed as a consequence of ischemia of the left side the initial deflection in all left ventricular leads becomes upright and the pattern of incomplete or complete LBBB develops

When the possibility of angina pectoris is suggested but unsettled by the history yet the routine resting electrocardiogram fails to demonstrate evidence of myocardial ischemia it may be possible to secure objective evidence of transitory ischemia by repeating the electrocardiogram immediately after a standard exercise test according to the method of Master or after the inhalation of a mixture of 10% oxygen and 90% nitrogen according to the method of Levy A positive stress test duplicates the changes in spontaneous angina pectoris although the usual abnormality is pathologic S-T depression described above an occasional but significant alternative is acute S T elevation of subepicardial injury reflecting ischemic changes extending diffusely through the wall

The Master two step technic is recommended for ambulatory patients because (1) amount of exercise can be adjusted to duplicate that which provoked pain or that encountered in the customary activity (2) the incidence of positive electrocardiographic findings is higher but the incidence of untoward reactions is lower than with the anoxia test After accurately marking all lead positions while the resting electrocardiogram is obtained all precordial and unipolar leads are taken from identical points as rapidly as possible after the stress concentrating on the leads most likely to show abnormalities If the resting electrocardiogram was abnormal the findings will predicate these leads If the original electrocardiogram was not definitely abnormal suspicion should be directed against leads with very slight S-T sagging or a long horizontal S T course The leads in which abnormalities are most prone to develop are V_4 V and V_6 The substitution of the anoxia for the exercise test is advisable in the evaluation of certain drugs When interpreting the results of each test it is necessary to bear in mind that exercise or induced anoxia often causes considerable reduction in the amplitude of the T wave in normal individuals and rarely causes a change from an upright to an inverted T wave in transitional or left precordial leads When the change from an upright to an inverted T wave

of the customary left-to-right septal vector is manifested by abnormal QS or QR complexes in overlying leads, *a reversal to a right-to-left septal vector is manifested by left bundle branch block* Abnormal Q waves or bundle branch block are not produced by lesions limited to the subepicardial myocardium, but may occasionally develop when pericarditis is merely one manifestation of a widespread myocarditis involving the subendocardial layer, as well Low voltage of QRS may be associated with both myocardial infarction and pericarditis particularly when *the latter is accompanied by effusion*

2) S-T segment and T waves The S-T elevation of acute myocardial infarction tends *to exceed that associated with pericarditis*, yet is more localized, just as the area of injury is more sharply demarcated Reciprocal depression is recorded in leads over portions of the ventricle opposite acute myocardial infarction, but is not found in pericarditis In subacute pericarditis, the T waves are commonly inverted in all precordial leads, whereas it is unusual to find such widespread inversion in myocardial *infarction Furthermore the inverted T waves of pericarditis* seldom attain the depth commonly seen in infarction and the evolution of the S-T changes is usually more rapid in pericarditis than in myocardial infarction In spite of the foregoing criteria pericarditis and myocardial infarction may be confused electrocardiographically Diagnostic difficulties may be encountered in the presence of the following patterns

   a) Localized abnormal elevation of the S-T junction and/or cove-plane inversion of the T wave in the presence of QRS complexes of normal or low voltage and normal contour and duration are characteristic of a lesion of the subepicardial layer of myocardium and may be attributable to pericarditis, or an infarct that is not interfering with response to the activating impulse Before reaching a diagnosis, it is necessary to consider the possibility of a pericardial reaction, secondary to a relatively small infarct localized to a portion of the wall not subtended by the 6 Wilson precordial and limb leads If a complete electrocardiogram was not taken, Leads V_7, V_8, and HV_2-HV_8, inclusive should be obtained to investigate the *possibility of a localized basal or posterior infarct Assuming that such has* been excluded the ST-T abnormalities may be the result of a localized subepicardial myocarditis or an infarct that is either confined to the subepicardial layer of myocardium or too patchy to obliterate response to the activating impulse In either event, an acute lesion is indicated by elevation in serial tracings, an old residue by fixation For further differentiation, the history will usually be needed

   b) Localized reduction in the amplitude of the R wave in a given lead below that attained in adjacent leads to the right and left Such a finding suggests patchy infarction but may be associated with pericardial thickening or effusion

c Chronic pericarditis The QRS complex is generally low in voltage but is not abnormal in contour or duration Localized reduction in the amplitude of the r wave may be observed in one or more leads, as compared with that in adjacent leads to right and left perhaps due to variations in pericardial thickening Significant Q wave patterns are absent The electrical position of the heart is generally fixed showing no appreciable change with body position or with respiration *Fixation of the electrical position* is suggestive of constrictive pericarditis if the heart is small or normal in size but has no significance if the heart is enlarged for the larger the heart the less the shift of electrical position with change of body position or with respiration The S-T junction is usually isoelectric The T waves are flat or inverted in leads facing the left ventricle and sometimes in right ventricular leads as well

evaluated On the other hand if localized subendocardial ischemia is the sole lesion leads facing the opposite wall may show a relatively slight S-T elevation Frequently, acute ischemia involves the entire circumference of the subendocardial layer of the left ventricle causing acute S-T depression in leads over the anterior, lateral and posterior walls

c Lead aV_R is the only routine lead that invariably reflects cavity potentials Acute subendocardial ischemia is manifested by transitory elevation of the S-T junction in Lead aV_R

B ACUTE DIFFUSE SUBENDOCARDIAL INJURY

1 QRS shows acute changes of one of the following types

a Complete or incomplete LBBB when the bundle branch or left side of the septum is involved This is transitory if recovery occurs, but may persist if the ischemia progresses to infarction

b Abnormal QR complexes characterized by a Q wave 03 sec or more in duration from onset to nadir and 25% or more of the amplitude of the succeeding R If the diffuse injury is transitory the abnormal QR complexes are replaced by normal patterns if it progresses to infarction the abnormal patterns persist

2 Q T interval – lengthened

3 S T and T waves – changes are similar in type to those in patchy injury but are greater in degree

C ACUTE SUBENDOCARDIAL INFARCTION discussed later

MYOCARDIAL INFARCTION

In the discussion of bundle branch block left ventricular hypertrophy right ventricular hypertrophy and dilatation pericardities and subendocardial injury some of the electrocardiographic features that may be mistaken for myocardial infarction were emphasized and the differential diagnosis was considered In the electrocardiographic diagnosis of myocardial infarction, the following three supplementary objectives should be kept in mind I Estimation of the distribution of the infarct between endocardium and epicardium, II Estimation of its size and location with reference to the cardiac surface and III Estimation of its age Although efforts to attain these objectives from the electrocardiographic findings alone are of academic interest the interpretation of the electrocardiogram in the light of clinical data is always advisable to minimize errors

I ELECTROCARDIOGRAPHIC ESTIMATION OF THE DISTRIBUTION OF AN INFARCT BETWEEN ENDOCARDIUM AND EPICARDIUM is based chiefly on QRS configuration but is aided by the S T pattern in leads from the left precordium axilla back or from the lower esophagus stomach or left leg When the infarct is large three concentric zones can be distinguished pathologically and usually upon electrocardiographic examination as well (A) a central zone of transmural infarction extending through the entire wall from endocardium to epicardium (B) a marginal zone of infarction confined to a portion of the wall almost always the subendocardial layer and (C) an outlying zone of ischemia manifested by pallor and absence of histologic evidence of degeneration If the infarct is small only the marginal and ischemic zones may be demonstrable

A FINDINGS IN LEADS SUBTENDING A CENTRAL ZONE OF TRANSMURAL INFARCTION

1 Characteristic findings are likely to be encountered when the septal vector maintains its normal left to right direction and a relatively large central zone of transmural infarction is present in the free wall The registration of an abnormal QS complex in a lead facing

represents a normal variant, the Q-T interval is usually shortened the change can usually be prevented by repetition of the test after ergotamine On the other hand neither the standard exercise nor the anoxia tests produce significant S-T depression in normals provided pseudodepression from tachycardia or exaggerated auricular T waves (page 120) are excluded Although the S-T junction may be 5 mm or more below the P-R junction pseudodepression is recognized by the continuity of the curves of the P-R and S-T segments and by the progressive ascent of the S-T segment in a concavely upward curve, beginning at the S-T junction

A ACUTE PATCHY SUBENDOCARDIAL INJURY (spontaneous or induced by exercise or anoxia test) Induced changes usually attain a maximum immediately after stress but may be delayed for a few minutes

1 QRS – usually no change

2 Q-T interval – transitory lengthening

3 S-T segment and T wave

a Leads facing the epicardial surface of the injured area The most significant change is a transitory horizontal or downwardly sagging depression of the S-T segment amounting to 5 mm or more This measurement is most easily made from the level of the T-P segment, instead it is made from the level of the P-R junction when the T-P segment is obliterated or the P-R junction is significantly depressed by a T_P wave (page 120) This is indicative of acute injury to the underlying subendocardial layer, provided pseudodepression (discussed above) and reciprocal depression from acute injury of the subepicardial aspect of the opposite wall (discussed below) are excluded Starting from its low take-off the abnormal S-T segment may pursue a horizontal course for a variable interval to end in an upright T wave or it may slope downward in a straight line in a concavely curved or rarely, in a convexly curved line to end in an inverted diphasic or upright T wave Reversal in the direction of the T wave is usually secondary to the primary and significant S-T displacement, changes in the direction of the T wave without alteration in the S-T segment are not diagnostic With acute subendocardial injury in the posterior wall previously inverted T waves in V_4 V_5 and V_6 may become transiently upright

The fact that the change in S-T segment and T wave as well as the depression of the S-T junction are the result of acute subendocardial ischemia and injury is verified by their abrupt appearance transient duration and prompt disappearance in serial tracings On the other hand, development of subendocardial infarction is associated with a much prolonged S-T evolution in serial tracings progression from a patchy to a diffuse subendocardial infarct may be marked by the appearance of QRS abnormalities as described below

The surface relations of the acutely ischemic area may be roughly mapped out from the leads showing the acute S-T depression e g localization to Leads V_4 V_5, and V_6 indicates ischemia of the anterolateral wall of the left ventricle, localization to Leads V_7 V_8 and aV_f indicates ischemia of the posterior wall localization to V_1 V_2 and V_3 points to ischemia of the septum Classical S-T abnormalities usually develop in a limited number of leads but are often accompanied by nondiagnostic changes in the S-T or T complexes in other leads Distribution of the lesion in a circular fashion around the entire inner circumference of the left ventricle including the left side of the septum is a common pathologic finding

b Leads facing the cardiac wall opposite the injured area Whenever acute S-T depression is observed a study of leads facing the opposite cardiac wall is essential before a final interpretation is made, because S-T depression simulating that due to subendocardial ischemia may be merely a reciprocal effect of acute infarction of the opposite wall In such an event leads facing the opposite wall show QRS abnormalities of infarction and a much greater S-T elevation than the depression which is being

evaluated On the other hand if localized subendocardial ischemia is the sole lesion leads facing the opposite wall may show a relatively slight S-T elevation Frequently acute ischemia involves the entire circumference of the subendocardial layer of the left ventricle causing acute S-T depression in leads over the anterior lateral, and posterior walls

c Lead aV_R is the only routine lead that invariably reflects cavity potentials Acute subendocardial ischemia is manifested by transitory elevation of the S-T junction in Lead aV_R

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3 S T and T waves — changes are similar in type to those in patchy injury but are greater in degree

C ACUTE SUBENDOCARDIAL INFARCTION - discussed later

MYOCARDIAL INFARCTION

In the discussion of bundle branch block, *left ventricular* hypertrophy right ventricular hypertrophy and dilatation pericarditis and subendocardial injury some of the electrocardiographic features that may be mistaken *for myocardial* infarction were emphasized and the differential diagnosis was considered In the *electrocardiographic* diagnosis of myocardial infarction the following three supplementary *objectives should* be kept in mind I Estimation of the distribution of the infarct between endocardium and epicardium II Estimation of its size and location with reference to the cardiac surface and III Estimation of its age Although efforts to attain these objectives from the electrocardiographic *findings alone are of* academic interest the interpretation of the electrocardiogram in the light *of clinical data is* always advisable to minimize errors

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A FINDINGS IN LEADS SUBTENDING A *CENTRAL* ZONE OF TRANSMURAL INFARCTION

1 Characteristic findings are *likely to* be encountered when the septal vector maintains its normal *left* to right direction and a relatively large central *zone* of transmural *infarction* is present in the free *wall* *The* registration *of an abnormal* QS complex in a lead *facing*

represents a normal variant the Q-T interval is usually shortened, the change can usually be prevented by *repetition of the test after ergotamine* *On the other hand* neither the standard exercise nor the anoxia tests produce significant S-T depression in normals, provided pseudodepression from tachycardia or exaggerated auricular T waves (page 120) are excluded *Although the S-T junction may be* .5 mm or more below the P-R junction pseudodepression is recognized by the continuity of the curves of the P-R and S-T segments and by the progressive ascent of the S-T segment in a concavely upward curve beginning at the S-T junction

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2 Q-T interval – transitory lengthening

3 S-T segment and T wave

a Leads facing the epicardial surface of the injured area The most significant change *is a transitory horizontal or downwardly sagging* depression of the S-T segment amounting to .5 mm or more This measurement is most easily made from the level of the T-P *segment, instead it is made from the level of the P-R junction when the* T-P segment is obliterated or the P-R junction is significantly depressed by a T_P wave (page 120) This is indicative of acute injury to the underlying subendocardial layer provided pseudodepression (discussed above) and reciprocal depression from acute injury of the subepicardial aspect of the opposite wall (discussed below) are excluded Starting from its low take-off the abnormal S-T segment may pursue a horizontal course for a variable interval to end in an upright T wave or it may slope downward *in a straight line* in a concavely curved or rarely in a convexly curved line to end in an inverted diphasic or upright T wave Reversal in the direction of the T wave *is usually secondary to the primary and significant S T displacement, changes in the* direction of the T wave without alteration in the S-T segment are not diagnostic With acute subendocardial injury in the posterior wall previously inverted T waves in V_4 V_5 and V_6 may become transiently upright

The fact that the change in S-T segment and T wave as well as the depression of the S-T junction are the result of acute subendocardial ischemia and injury is verified by their abrupt appearance transient duration and prompt disappearance in serial tracings *On the other hand development of subendocardial infarction is associated* with a much prolonged S-T evolution in serial tracings progression from a patchy to a diffuse subendocardial infarct may be marked by the appearance of QRS abnormalities as described below

The surface relations of the acutely ischemic area may be roughly mapped out from the leads showing the acute S-T depression, e g localization to Leads V_4 V_5 and V_6 indicates ischemia of the anterolateral wall of the left ventricle, localization to Leads V_7 V_8 and aV_F indicates ischemia of the *posterior wall localization to* V_1 V_2 *and* V_3 points to ischemia of the septum Classical S-T abnormalities usually develop in a limited *number of leads* but are often accompanied by nondiagnostic changes in the S-T or T complexes in other leads Distribution of the lesion in a circular fashion around the entire inner circumference of the left ventricle including the left side of the septum is a common pathologic finding

b Leads facing the cardiac wall opposite the injured area Whenever acute S T depression is observed a study of leads facing the opposite cardiac wall is essential before a final interpretation is made because S-T depression simulating that due to subendocardial ischemia may be merely a reciprocal *effect of acute infarction of the opposite wall* In such an event leads facing the opposite wall show QRS abnormalities of infarction and a much greater *S-T elevation than the depression* which is being

b

familiar finding in spontaneous or induced angina pectoris and may be present in electrocardiograms obtained shortly after the onset of attacks that eventuate in myocardial infarction Repetition of the tracing after a few hours should disclose the development of abnormal QS or QR complexes if infarction is present

c Patchy ... al de- ... will depend upon the algebraic summation of positive potentials derived from activation of the preserved islands and negative cavity potentials transmitted through the dead muscle An initial upstroke may be recorded if the preserved islands are relatively large or if the cavity potentials are reduced by infarction of the opposite wall

B FINDINGS IN LEADS SUBTENDING A MARGINAL ZONE OF SUBENDOCARDIAL INFARCTION

1 Characteristic findings expected when the septal vector is in the normal left-to-right direction and a marginal zone of sufficient size is present

a QR or Qr patterns ... stroke ... Abnorm... 03 sec... succeed... of subendocardial infarction Such patterns are recorded over diffuse infarcts limited to the subendocardial layer and at the margins of a transmural infarct due to the tendency for large infarcts to extend farther on their endocardial than on their epicardial surface A rough estimate of the relative thickness of the infarcted subendocardial and living subepicardial layers may be made from the time of onset to nadir of the Q as compared with the time from onset to peak of R and from the relative amplitudes of the Q and R waves The state of the living subepicardial layer is reflected in the S T segments and T waves

1) Acute injury to subepicardial muscle is manifested by Q-T prolongation elevation of the S T segment with monophasic upright T wave The S-T and T patterns have been described on page 141 The upward displacement of the S ... nt is likely to be greater in inf... d is more likely to be limited ... left ventricular leads ... to leave residual abnormalities in repolarization is accompanied by progressive return of the S-T junction towards the isoelectric line together with increasing cove-plane inversion of the T waves gradual recovery of normal repolarization is marked by progressive decrease in the depth of the inverted T waves and eventual replacement by normal upright T waves The evolution is usually much slower in infarction than in other types of subepicardial injury

The decision as to whether or not the recovered muscle is ischemic is based more on the response to the exercise or anoxia test rather than on the S-T and T patterns in a tracing taken at rest These tests are contraindicated during the first few weeks after myocardial infarction but are of help in estimation of the adequacy of the collateral circulation after clinical recovery from the acute episode has occurred They are helpful in estimating the amount of activity to be advised and in making a prognosis The criteria for the evaluation of the response to these tests have been given on page 143

the epicardial surface of the left ventricle * which should normally exhibit an R wave as the major deflection constitutes evidence of transmural infarction of the underlying wall In the occasional case in which the entire thickness of the subjacent myocardium is dead, the QS deflection has smooth descending and ascending limbs the T wave resembles that in leads from the left ventricular cavity and remains fixed in serial tracings QS complexes with fixed ST-T patterns in one or more left ventricular leads are significant of acute transmural necrosis when neighboring leads show serial ST-T changes typical of injury or organization Such QRS-T complexes are derived principally from the opposite uninfarcted wall and are roughly the reciprocal of patterns that can be recorded from chest leads facing the epicardial surface of this wall

In the event of islands of acutely injured but living, muscle in the more superficial layers of the transmural infarct the QS deflection is usually notched or slurred from the presence of an embryonic r wave, the Q-T interval is prolonged the S-T complex is at first markedly elevated and merged with a tall upright T wave whereas in subsequent serial tracings the S-T junction gradually approaches the isoelectric line, and the T waves undergo progressive cove-plane inversion

Lead aV_R reflects the potential variations of the cavity as a whole and thus will exhibit serial ST-T changes from injured or organizing muscle in some segments of the wall even in the presence of a central zone of transmural necrosis If the central zone is small semidirect chest leads may fail to show a QS deflection but instead may show an abnormal QR complex referable to the surrounding marginal zone of subendocardial infarction

2 *Absence of abnormal QS or QR patterns may be encountered despite the presence of transmural or subendocardial infarction* When the clinical findings suggest infarction but the electrocardiogram yields no confirmatory evidence the first step is to determine whether or not sufficient leads have been taken to explore adequately the left ventricle This is especially important when the transitional zone has been displaced into the mid-axilla (as a result of right ventricular dilatation and clockwise rotation) for under these circumstances, additional back leads may be needed to cover the left ventricle Even though the electrode subtends a central zone of transmural infarction abnormal QS or QR patterns may not be registered for one of the following reasons

a Septal vector directed from right to left as in complete and incomplete left bundle branch block Activation of the septum by impulses passing from right to left causes early positivity of the left ventricular cavity and thus an initial upstroke in all leads facing the epicardial surface of the left ventricle irrespective of the anatomic condition of the free wall In leads over intact portions of the outer wall this initial upstroke of septal origin is followed by a second and often larger upstroke coming from activation of the free wall to produce a pattern of complete or incomplete LBBB depending upon the duration of the QRS interval In leads over a transmurally infarcted section of the outer wall the initial r of septal origin may be similar but the final component is diametrically opposite consisting of a broad S instead of the late R To draw inferences from the registration of such an rS complex in LBBB one must be certain that the leads in which it is recorded are dominated by the potential variations of the left and not the right ventricle since an rS is the expected finding in right ventricular leads in patients with LBBB

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c Patchy infarction with preserved islands of muscle in the subendocardial layer or scattered through the wall Under these circumstances, the direction of the initial deflection and magnitude of any r wave recorded in an overlying precordial lead will depend upon the algebraic summation of positive potentials derived from activation of the preserved islands and negative cavity potentials transmitted through the dead muscle An initial upstroke may be recorded if the preserved islands are relatively large or if the cavity potentials are reduced by infarction of the opposite wall

B FINDINGS IN LEADS SUBTENDING A MARGINAL ZONE OF SUBENDOCARDIAL INFARCTION

1 Characteristic findings expected when the septal vector is in the normal left-to-right direction and a marginal zone of sufficient size is present

a QR or Qr patterns recognized as abnormal because of increased duration of the downstroke of the Q and increased amplitude of the Q in reference to the succeeding R Abnormal QR patterns in left ventricular leads characterized by an initial downstroke 03 sec or longer from onset to nadir and more than 25% of the amplitude of the succeeding R wave are diagnostic of subendocardial infarction Such patterns are recorded over diffuse infarcts limited to the subendocardial layer and at the margins of a transmural infarct due to the tendency for large infarcts to extend farther on their endocardial than on their epicardial surface A rough estimate of the relative thickness of the infarcted subendocardial and living subepicardial layers may be made from the time of onset to nadir of the Q as compared with the time from onset to peak of R and from the relative amplitudes of the Q and R waves The state of the living subepicardial layer is reflected in the S-T segments and T waves

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2 Absence of abnormal QS or QR patterns may be encountered *despite the presence of* transmural or subendocardial infarction When the clinical findings suggest infarction but the electrocardiogram yields no confirmatory evidence the first step is to determine whether or not sufficient leads have been taken to explore adequately the left ventricle This is especially important when the transitional zone has been displaced into the mid-axilla (as a result of right ventricular dilatation and clockwise rotation) *for under these* circumstances additional back leads may be needed to cover the left ventricle Even *though the electrode subtends a central zone of transmural infarction* abnormal QS or QR patterns may not be registered for one of the following reasons

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[illegible] (or transmural) [illegible] The [illegible] S-T [illegible] with [illegible] of the [illegible] of [illegible]

2 [illegible] [illegible] The [illegible]

E FINDINGS IN LEADS [illegible] WALL OPPOSITE AN INFARCT. Leads over the [illegible] will tend to show patterns [illegible] facing the [illegible] of the R wave, [illegible] depression of the S-T junction and [illegible] together with [illegible] of the erect T wave. These [illegible] are of particular value in the detection of [illegible] of the posterior wall usually [illegible] changes in Lead aV [illegible] or V_1 but are [illegible] changes in [illegible] leads. Exaggerated R and T waves and depressed S-T segments in precordial leads should always arouse the suspicion of [illegible] and prompt one to take Leads [illegible], HV [illegible] and HV [illegible] which should reveal diagnostic signs of posterobasal infarction if present.

II. ESTIMATION OF THE SIZE AND LOCATION OF THE INFARCT WITH REFERENCE TO THE CARDIAC SURFACE is based on the fact that tracings obtained through multiple chest leads reflect chiefly the potential variations of the epicardial surface subtended by the exploring electrode and is made by mapping out the anatomic relationship of leads showing abnormal QS or QR patterns to the surface of the heart. Such estimates presuppose the existence of the normal left to right septal vector and the consequent registration of abnormal QS or QR deflections in electrode positions overlying central and marginal zones of infarct. In the presence of LBBB the size of the infarct cannot be judged from the QRS pattern. The development of the conduction defect along with S-T patterns typical of injury may be confirmative of infarction but only a rough approximation of its distribution and size in the free wall may be ventured from the leads showing typical serial changes in ST-T complex. If abnormal QS or QR complexes are present overestimation of the area of infarction is likely when the chest is small and the electrode positions close together or when the heart is small; underestimation occurs when the chest or heart is large. In serial tracings during the acute stage the apparent size of the infarct may decrease through recovery in the marginal zone or may increase as a result of extension.

Satisfactory correlation between the electrocardiographic estimate of the size and position of the infarct and the pathologic findings can be achieved provided a sufficient number of semi-direct leads is available to cover the surface of the heart and to demarcate the zones of reference of the potential variations of the two ventricles. A prerequisite to the interpretation of multiple chest leads consists in the delineation of the transitional zone between the two ventricles in the lower and upper precordium and preferably in the back as well.

The transitional zone serves as an index of the projection of the anterior and posterior ends of the septum onto the chest wall and thus serves as a dividing line between the portion of the chest receiving predominantly the potential variations of the left ventricle and the portion receiving chiefly the potential variations of the right ventricle. When the transitional zone is in its usual position in the vicinity of Lead V_3 anteriorly and near the mid-line posteriorly, the potential variations recorded through precordial leads at and to the left of the transitional zone (V_3 and V_4) come chiefly from the apical portion of the anterior wall of the left ventricle; those obtained in HV_4 come chiefly from the basal portion of the anterior wall of the left ventricle; those registered through the usual axillary leads (V_5, V_6 and V_7) come mainly from the anterolateral, lateral, and posterolateral aspects of the left apex respectively; those in HV_5, HV_6 and HV [illegible] from the corresponding portions of the base; those recorded in mid-back Leads HV_8 and V_8 and in the low esophageal leads usually come chiefly from the posterobasal wall of the left ventricle; those in leads from the left leg (AV_F), stomach, and lower part of the back

2) Preservation of the overlying subepicardial layer in acute subendocardial infarction permits registration of effects of acute subendocardial injury namely, abnormal sagging or horizontal depression of the S-T junction in overlying leads, with progressive changes in serial tracings These may consist of increased downwar S-T displacement in the event of exacerbation in subendocardial injury or the replacement by electrocardiographic signs of subepicardial injury if the lesion extends centrifugally On the other hand subsidence is manifested by gradual return of the S-T segment to the isoelectric line Residual delay in repolarization o the subendocardial layer may result in broad upright T waves with prolonged Q T interval

b qR patterns, recognized as abnormal because of distinct notching of the upstroke of the R wave When there is an antecedent q wave, notching of the ascending limb of tl R is referable to a defect in conduction in the outer wall of the left ventricle and may be correlated with healed patchy infarction at autopsy The combination of a distinct q wave with notching of the upstroke of the R wave may be correlated with a healed subendocardial infarct that extends in patchy fashion into the more superficial layers On the other hand, notching of the upstroke of an initial R wave is of no significance in the diagnosis of infarction and may be observed as a normal variant in leads near the transitional zone and as a manifestation of uncomplicated LBBB in any left ventricular lead

2 Absence of characteristic findings

a A qR complex consistent with that recorded in normals or in uncomplicated LVH may be found if the layer of subendocardial infarction is thin in proportion to the overlying intact muscle or with coexistent infarction of equivalent thickness in the subendocardial layer of opposite walls of the left ventricle Even though the duration of the q wav and qR ratio are within normal limits a presumptive diagnosis of a thin infarct of the subendocardial aspect of the anteroseptal wall of the left ventricle may sometimes be made on the basis of a reversal in the usual relationships in successive leads taken to the left of the transitional zone In normals and in uncomplicated LVH the expected finding is a change from an Rs to a qR pattern with slight increase in duration of q and in qR ratio as the electrode is moved from positions over the anteroseptal wall to the lateral and then the posterior wall When the duration of the q and the qR ratio are maximal in a lead over the anteroseptal wall of the left ventricle beyond the transitional zone and dwindle as the electrode is moved leftward a presumptive diagnosis of a thin infarct of the subendocardial aspect of the anteroseptal wall may be made

b The registration of an initial upstroke in place of a QR deflection may occur for the same reasons as in transmural infarction discussed on page 146

C FINDINGS IN LEADS SUBTENDING A MARGINAL ZONE OF SUBEPICARDIAL INFARCTION — discussed on page 143

D FINDINGS IN LEADS SUBTENDING AN OUTLYING ZONE OF ISCHEMIA

1 Subepicardial ischemia (or transmural ischemia) Even though the ischemia is transmural the S-T patterns recorded in an overlying chest lead reflect the changes in the subepicardial layer and resemble the pattern associated with ischemia (or injury) limited to the subepicardial layer There is transient elevation and straightening of the S-T segment with monophasic upright T, followed by rapid return of the S-T junction to the isoelectric line accompanied by cove-plane inversion of the T wave The QRS pattern remains normal in serial tracings as long as the ischemic area does not become infarcted, whereas abnormal Q waves appear in the event of extension of the infarct into a previously ischemic area After clinical recovery the presence or absence of residual

subepicardial (or transmural) ischemia is judged from the exercise or anoxia test The development of transient S T elevation with prompt return to the control pattern is indicative of residual subepicardial (or transmural) ischemia

2 Subendocardial ischemia Evidence of subendocardial ischemia is recorded in leads facing the epicardial surface only when the intervening subepicardial muscle is intact The electrocardiographic features have been described on page 144

E FINDINGS IN LEADS OVER THE UNINVOLVED WALL OPPOSITE AN INFARCT Leads over the opposite intact wall tend to show patterns reciprocal to those recorded in leads facing the infarct namely exaggeration of the R wave initial depression of the S-T junction and progressive return to the isoelectric line together with increasing height of the erect T wave These reciprocal effects appearing in precordial leads are of particular value in the detection of posterobasal infarction Infarcts confined to the basal third of the posterior wall usually produce no diagnostic changes in Lead aV_F and sometimes not in V_7 or V_8 but are likely to cause reciprocal changes in customary or high precordial leads Exaggerated R and T waves and depressed S T segments in precordial leads should always arouse the suspicion of posterobasal infarction and prompt one to take Leads HV_6 HV_7, and and HV_8 which should reveal diagnostic signs if posterobasal infarction is present

II. ESTIMATION OF THE SIZE AND LOCATION OF THE INFARCT WITH REFERENCE TO THE CARDIAC SURFACE is based on the fact that tracings obtained through multiple chest leads represent chiefly the potential variations of the epicardial surface subtended by the exploring electrode and is made by mapping out the anatomic relationship of leads showing abnormal QS or QR patterns to the surface of the heart Such estimates presuppose the existence of the normal left to right septal vector and the consequent registration of abnormal QS or QR deflections in electrode positions overlying central and marginal zones of infarct In the presence of LBBB the size of the infarct cannot be judged from the QRS pattern The development of the conduction defect along with S T patterns typical of injury may be confirmatory of infarction but only a rough approximation of its distribution and size in the free wall may be ventured from the leads showing typical serial changes in ST-T complex If abnormal QS or QR complexes are present overestimation of the area of infarction is likely when the chest is small and the electrode positions close together or when the heart is small underestimation occurs when the chest or heart is large In serial tracings during the acute stage the apparent size of the infarct may decrease through recovery in the marginal zone or may increase as a result of extension

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The transitional zone serves as an index of the projection of the anterior and posterior ends of the septum onto the chest wall and thus serves as a dividing line between the portion of the chest receiving predominantly the potential variations of the left ventricle and the portion receiving chiefly the potential variations of the right ventricle When the transitional zone is in its usual position in the vicinity of Lead V_3 anteriorly and near the mid-line posteriorly the potential variations recorded through precordial leads at and to the left of the transitional zone (V_3 and V) come chiefly from the apical portion of the anterior wall of the left ventricle those obtained in HV_4 come chiefly from the basal portion of the anterior wall of the left ventricle those re istered through the usual axillary leads (V_5 V_6 and V_7) come mainly from the anterolateral lateral and posterolateral aspects of the left apex respectively those in HV_5 HV and HV_7 from the corresponding portions of the base those recorded in mid back Leads HV_8 and V_8 and in the low esophageal leads usually come chiefly from the posterobasal wall of the left ventricle those in leads from the left leg (AV_F) stomach and lower part of the back

—

(LV_7 and LV_8) come principally from the diaphragmatic surface The potential variations of the septum have a relatively greater influence on the findings in leads to the right usually V_1 and V_2 When the transitional zone is displaced to the left of the mid-clavicular line, as a result of dilatation of the right ventricle and/or clockwise rotation of the heart, the potential variations of the anterior wall of the left ventricle are referred to the anterior axilla, those of the lateral wall to the posterior axilla The following description of typical electrocardiographic abnormalities associated with the commoner sites of infarction is based on the assumption of normal cardiac position and would have to be modified as described above, in the event of displacement of the transitional zone

A INFARCTION OF THE ANTERIOR WALL OF THE LEFT VENTRICLE The usual large infarct involves the apical third to two-thirds of the anterior wall continues into the adjoining septum and into the apical third or more of the lateral wall The electrocardiographic manifestations of the septal and lateral portions will be reviewed insofar as they contribute to the diagnosis of anterior infarction but will be elaborated upon separately below Large anterior infarcts usually have a central zone of sufficient size to permit the registration of QS patterns in one to three overlying leads, usually V_3 V_4, and/or V_5 These QS patterns are recognized as abnormal and referable to transmural infarction from the findings in leads facing the right side of the septum and right ventricle, considered together with findings in leads over surrounding portions of the left ventricle

If the QS complexes are an expression of transmural infarction leads over adjoining portions of the left ventricle laterally or above should display abnormal QR deflections owing to the fact that a central zone of transmural infarction is almost always surrounded pathologically by a marginal zone of subendocardial infarction The QR complex is classed as abnormal because of either (1) a Q wave 03 sec or more in duration and more than 25 per cent of the amplitude of the R wave, or (2) a q wave of normal duration that is followed by a notched or coarsely slurred upstroke Registration in HV_3 HV_4, and/or HV_5 is a manifestation of a marginal zone in the base of the anterior wall registration in V_5 V_6 and/or V_7 is the result of a marginal zone in the anterolateral portion of the apex

The presence of diagnostic evidence of septal infarction in V_{3R} V_1 and V_2 confirms the interpretation of a QS complex in V_3 as a manifestation of infarction but may open the question as to whether the QS in V_3 is a manifestation of a transmural infarction of the anterior wall of the left ventricle an infarct of the septum or both The presence of a normal rS complex in V_{3R} and V_1 indicates that a QS deflection in V_3 is a manifestation of transmural anterior infarction provided that abnormal QR complexes are recorded in leads above and/or to the left of V_3 This latter provision is necessary to distinguish right ventricular dilatation, which may be manifested by rS patterns in V_{3R} V_1 and V_2, true or pseudo QS complexes in one or more leads near the transitional zone (such as V_3 V_4 or V_5) but normal qR patterns in leads over the left ventricle both at the customary and high levels

Large anterior infarcts also continue around the tip of the left ventricle into the apical portion of the posterior wall Extensions limited to the apical third of the posterior wall are seldom recognizable in the usual chest and limb leads, extentions into the apical half or more of the posterior wall are generally manifested by abnormal QR patterns in Lead aV_F and in leads from the back below the diaphragm

With smaller anterior infarcts the central zone may be absent or too small to permit the registration of a QS deflection in any semidirect lead Overlying leads should exhibit abnormal QR complexes referable to infarction of the subendocardial layer and preservation of some subepicardial muscle Small infarcts limited to the apical third of the anterior wall of the left ventricle are manifested by abnormal QR patterns localized to Leads V_3 V_4 and/or V_5, the relatively rare small infarcts confined to the basal portion of the anterior wall are manifested by abnormal QR patterns in HV_3 HV_4 and/or HV_5

Patchy infarction or smaller areas of confluent infarctions may not have a sufficiently extensive marginal zone to permit the registration of abnormal QR patterns but may nevertheless, exhibit typical serial S-T-T evolution The diagnosis is established from the

history together with the finding of normal qR complexes and typical ST-T evolution in serial tracings after exclusion of a *large infarct* which has extended into the septum sufficiently to produce left bundle branch *block* and therefore, initial R waves to replace the normal q, in all left ventricular leads

B INFARCTION OF THE SEPTUM occurs as a part of practically all large anterior *infarcts* of most posterior infarcts but not of primary lateral infarcts Furthermore, the infarct may be primary in the septum, but seldom remains confined to *this structure* usually *extending into* the anterior wall of the left ventricle, less commonly into the posterior wall The electrocardiographic manifestations of the accompanying infarction of the anterior or posterior wall are described separately The association of auriculoventricular *block* with signs of posterior (or rarely anterior) infarction suggests extension into the septum *and atrioventricular node* Continuation of the anterior or posterior infarct across the septum *into the adjoining wall of* the right ventricle does not produce electrocardiographic signs *referable to the right ventricular* involvement Infarction of the septum may not produce QRS abnormalities particularly after healing or may be manifested by one of the following patterns

1 Right bundle branch block may be produced by septal infarct irrespective as to whether or not the lesion continues into the adjoining free wall of the right ventricle

   a A diagnostic QR pattern characterized by a QRS interval of 12 sec or more an abnormal Q wave and a prominent late R wave in right precordial leads may result from extensive infarction of the septum involving the apical half or more pathologically Continuation of the infarct into the anteroseptal wall of the left ventricle is manifested by the *registration* of an abnormal Q wave preceding the expected rS in leads to the left of the transitional *zone* Recent infarction is indicated by typical ST-T evolution in the right *precordium* and in contiguous leads over the left ventricle

   b An alternative rSR' complex may rarely be registered as a variant of the diagnostic QR pattern and has been correlated with extensive infarction of the septum and *free* wall of the left ventricle The initial r represents positive potentials produced by activation of the uninfarcted portion of the septum and registered in right precordial leads because of the reduction of opposing negative potentials by extensive infarction of the outer wall of the left ventricle The diagnosis is verified in recent and *organizing* infarcts by typical serial ST-T changes

2 Left bundle branch block from septal infarction The presence of left bundle branch *block is established* from the following criteria (a) supraventricular origin of the impulse and transmission *through the* A-V node (b) prolongation of the QRS (12 sec or more with complete LBBB 10-11 sec with *incomplete*) (c) abnormally late intrinsicoid deflection in left ventricular leads (08 sec or *more in* complete LBBB 06-07 sec in incomplete LBBB) (d) initial upstroke in all left ventricular leads *The QRS pattern* of LBBB with *coexistent* infarction may be indistinguishable from that of uncomplicated LBBB *If the infarct* is recent, a diagnosis *from* the history may be confirmed by typical serial *ST-T evolution* At *times* the presence of infarction of the free wall can be inferred *from a marked reduction in* the R' *component* or its replacement by an S wave found localized *to* the leads facing the epicardial surface of the infarct These leads like all *others facing the free wall of the* left ventricle will show an initial R due to septal activation in a right-to left *vector* however the *R'* component over an infarcted outer wall should *be diminutive or replaced by an S derived from simultaneous* activation of the opposite uninfarcted wall Under the following circumstances even *more* distinctive QRS patterns *may be associated* with coexistent LBBB and infarction

   a LBBB with Q waves in left ventricular leads due to massive infarction of the septum In most cases of LBBB associated *with infarction* of a portion of the septum, the activation of the remainder of the septum *from* right to left produces sufficient positivity in the cavity of the left *ventricle* and at its epicardial *surface* to account for the initial

R wave in overlying precordial leads When the area of septal infarction is large negative potentials, derived from forces associated with activation of the right ventricle and transmitted through the right ventricular cavity and septal infarct, may preponderate and lead to the registration of a Q wave in left ventricular leads This Q is abnormal because of delay in activation of the free wall of the left ventricle and is followed by an abnormally late R from activation of living subepicardial muscle The abnormal QR deflection formed in this manner is considered relatively rare from post-mortem experience in comparison with the abnormal QR recorded in left ventricular leads in association with infarction of the subendocardial portion of the free wall (Fig 13)

b LBBB with infarction of the apical portion of the septum and abnormal Q waves in high precordial leads from coexistent infarction of the base of the outer wall Whenever all leads facing the apical portion of the left ventricle reveal the pattern of LBBB, the high precordial leads must be checked for direction of the vector at the base of the septum If an initial R wave is present in these leads, as well the diagnosis of LBBB is established and the presence of coexisting infarction may be suspected when (1) the R' component is diminutive or replaced by an S wave (2) typical serial ST-T changes are found When a distinct initial downstroke is present in high chest leads in association with the findings of LBBB in the customary chest leads, the interpretation of the Q wave depends upon its duration amplitude, and distribution Q waves of 03 sec or longer and more than 25% of the succeeding R waves are indicative of infarction of the subendocardial portion of the base, provided that a transitional effect is excluded When uncomplicated LBBB is manifested by QS complexes in right ventricular leads along with monophasic R waves in left ventricular leads QR complexes may be found in transitional leads as the result of an admixture Such QR complexes can be recognized as a transitional phenomenon by their relatively low voltage and limitation to one or two leads To justify the diagnosis of infarction abnormal QR complexes should be present in three or more leads To settle the diagnosis additional intervening leads may be needed

3 *Patterns in right precordial leads indicative of septal infarction in the absence of bundle branch block*

a qrS complexes of less than 12 sec point to septal infarction provided due consideration is given to right ventricular hypertrophy and incomplete left bundle branch block In the presence of a right-to-left septal vector qrS complexes may be recorded in leads over the body of a dilated right ventricle, the diagnosis of hypertrophy of the tricuspid ring is established by the presence of abnormal qR deflections in Leads V_{3R} V_{4R} or V_{5R} After exclusion of right ventricular hypertrophy it is necessary to determine whether the qrS is the result of septal infarction or merely a manifestation of incomplete left bundle branch block In either case the q comes from septal activation in a right-to-left direction the r from passage of the impulse through the outer wall of the right ventricle and the S from activation of the left ventricle The decision is based upon QRS contour in leads over the left ventricle The continuation of the q wave into left ventricular leads indicates that the qrS of right precordial leads was the result of septal infarction A consistent initial upstroke in all left ventricular leads indicates incomplete LBBB the presence or absence of coexisting infarction is based upon other findings described above

b QS deflections of less than 12 sec in all right ventricular leads also bring up the differential diagnoses between septal infarction without conduction defect and incomplete left bundle branch block The significant points in the analysis have been discussed above in cases with right ventricular qrS complexes If there is recent septal infarction the diagnosis may be confirmed by typical S-T evolution in serial records of V_1 and V_2

C INFARCTION OF THE LATERAL WALL OF THE LEFT VENTRICLE The commonest site in the lateral wall is the apical one third but most infarcts in this area are extensions of anteroapical lesions some are continuations of high lateral infarcts a few represent extensions of posterior infarcts and a few are primary in the apical portion of the lateral wall Infarction confined to this area may be manifested by abnormal QR patterns in Leads V_5 and/or V_6 only, infarction extending into this area from other portions of the heart is manifested by abnormal QRS patterns in additional leads depending upon the distribution of the remainder of the lesion The most common primary infarct of the lateral wall takes the form of a truncated cove with base near the atrioventricular groove Large infarcts of this type project sufficiently into the apical third of the lateral wall to produce abnormal QR patterns in V_5 V_6 and/or V_7 smaller infarcts confined to the basal half of the lateral wall are not detectable in these leads Signs suggestive of high lateral infarction may be present in Lead aV_l but the diagnosis is established by abnormal QR complexes (with or without QS patterns) in high axillary leads (HV_5 HV_6, and HV_7) Before QS deflections in high precordial and axillary leads are attributed to lateral infarction caution must be taken to exclude the possibility that they represent the potential variations of the right ventricle and/or cavity transmitted to the upper precordium and axilla because of vertical position of the heart In this event an rS [illegible] y to a narrow QS in leads further to the [illegible] responsible for the r wave In cases of right [illegible] ross the transitional zone additional leads over the left ventricle will be needed to clarify the diagnosis

D INFARCTION OF THE POSTERIOR WALL OF THE LEFT VENTRICLE is more likely to be missed electrocardiographically than infarction of the anterior or lateral walls because the posterior wall is less accessible to exploration by surface leads yet more subject to variations in anatomic relations as a result of the influence of the height of the diaphragm A portion of the posterior wall of one or both ventricles rests on the diaphragm the remainder faces towards the back of the thorax The portion resting upon the diaphragm depends upon the position of the diaphragm and heart With elevation of the diaphragm and a horizontal electrical position of the heart the potential variations of the right side of the septum and the posterior wall of the right ventricle are transmitted downward to dominate the tracing in aV_F and in subdiaphragmatic leads whereas the potential variations of the left side of the septum and posterior wall of the left ventricle are referred posteriorly and upward to dominate the pattern in lower esophageal leads high back Leads HV_7 and HV_8 and usually mid back Lead V_8 When the diaphragm is normal to low in position the posteroapical half or more of the left ventricle is in apposition and has the predominant influence on the findings in aV_F and other subidaphragmatic leads Under these circumstances the recordings from esophagus and back at the cardiac level represent potential variations transmitted in part from the posterobasal surface of the left ventricle in part through the mitral orifice and left atrium Lead V_8 may be utilized for evaluation of the potential variations of the posterobasal wall but Lead HV_8 represents too great an admixture to be dependable particularly in tracings containing a negative or diphasic P wave

The most common primary infarct of the posterior wall takes the form of a truncated cone with base parallel to the atrioventricular groove If the heart is in horizontal position a large posterior infarct may be undetectable in aV_F and in leads from the low back and stomach unless it extends sufficiently into the septum to produce typical signs of septal infarction (page 151) in these leads Recognition of infarction of the posterior wall of the left ventricle in such cases must be based upon the patterns in HV_7 HV_8 and V_7 V_8 but a clue as to its presence may be afforded by the reciprocal effects in anterior leads Smaller infarcts limited to the basal third of the posterior wall of the left ventricle may produce abnormal QR patterns confined to HV_7 HV_8 V_8 and esophageal leads just below the atrioventricular groove but not in leads from the stomach low back (LV_7 LV_8) or left leg (aV_F) regardless of the position of the heart

If the heart is in intermediate to vertical position posterobasal or posteroapical infarcts large enough to involve the middle third of the posterior wall should be manifested by *abnormal QR patterns in* aV_F The Q component of a QR complex is abnormal if its duration from onset to nadir is 03 sec or more and if more than 25% of the amplitude of the succeeding R wave provided the voltage in aV_F is 5 mv or more Even *in the presence of a* large central zone, the registration of QS deflections is unusual in leads facing an infarcted posterior left ventricular wall because the larger area subtended by the electrode permits more admixture of potentials from outlying marginal areas If there is an apparent QS in aV_F, careful examination of the tracing or of a repeat taken during deep breathing should reveal a late embryonic or actual r and should thus *identify the presence of a Qr complex* significant of infarction Such a study is necessary to exclude the normal QS complex found in some cases of horizontal position, as a result of fading of potentials that would have produced an initial r wave in leads less distant from the heart If some phase of breathing does not permit the recording of a normal rS complex the fact that the QS is a normal variant is *suggested* by its brevity and is *established by the fact that the time from beginning* to end is significantly less than the time from onset to termination of an rS complex in a right precordial lead

Complete auriculoventricular block may accompany massive septal extensions of posterobasal infarction Secondary auricular infarction may be manifested by auricular arrhythmias *but seldom by diagnostic changes in the P or* T_P *waves Extension of massive in*farcts across the septum into the posterior wall of the right ventricle is fairly common but does not significantly alter the electrocardiographic findings

Continuation of posterior infarcts into the basal portion of the lateral wall is manifested by abnormal QR patterns in HV_7 and HV_6, continuation into the apical portion of the lateral *wall by diagnostic signs in customary* V_7 *and* V_6 *Infarcts involving the apical but not the* basal portion of the posterior wall are usually continuations of anterior infarcts that involve the apical portion of the anterior wall, the septum and the posterior wall The ECG usually displays evidence of the anterior and sometimes the septal portion of the infarct but not the posterior unless it involves more than the apical third of the posterior wall The term *anteroposterior infarction is applied when the ECG shows evidence of simultaneous rather* than consecutive infarction

III *ELECTROCARDIOGRAPHIC ESTIMATION OF THE AGE OF THE INFARCT is based chiefly* on a comparison of the S-T pattern in serial tracings Recent infarction is manifested by progressive changes in S-T segment and T waves, healed infarction by a fixed pattern in serial tracings Serial changes in the degree of S-T displacement indicate the presence of injured myocardium that is still living and is destined either to death or recovery Subsequent serial *changes in the T wave have a similar connotation*

In recent infarction with acute subepicardial injury the S-T junctions show abnormal elevation that changes from day to day increasing if injury spreads receding towards the isoelectric line as it subsides the T waves are at first tall and monophasic upright then show increasing inversion of their terminal portions With the disappearance of acute injury the S-T junction becomes stabilized usually at the isoelectric level, and as organization proceeds the T waves at first show increasing cove-plane inversion (waxing phase) then a much more gradual decrease in depth (waning phase), and finally may return to a normal upright contour Permanent fixed S-T elevation and cove-plane inversion of the T waves associated with abnormal QS or QR patterns occurs with healed infarcts that involve a sufficiently large area of the wall to form ventricular aneurysms

The QRS pattern is of less help in determining age and often remains constant in serial tracings, even though the infarct is recent or organizing However significant changes in QRS pattern found when adequate technical precautions are taken to insure against variations in electrode position constitute evidence of activity A definite increase in duration and amplitude of the Q wave at the expense of the R wave at a constant electrode position would

indicate spread of an underlying subendocardial infarct towards the epicardium change from normal QRS complexes at the boundaries of the lesion to abnormal QR deflections would indicate increase in area of infarction On the other hand, abnormal QS and QR patterns found early in the stage of injury may show considerable increase in the R wave at the expense of the Q if a portion of the injured myocardium recovers Incomplete left bundle branch block is a common finding in recent infarction involving the septum but is often not permanent Upon its disappearance a left-to-right septal vector becomes predominant permitting the registration of Q waves in leads over the left ventricle The distribution of the infarct in its free wall may then be estimated from the duration and amplitude of these Q waves in comparison with the succeeding R waves

DRUGS CAUSING SPECIFIC ELECTROCARDIOGRAPHIC CHANGES

I. DIGITALIS AND RELATED CARDIAC GLYCOSIDES

A QRS complex is not altered by therapeutic doses Gross overdosage may cause notching, slurring or prolongation

B Q T interval is progressively shortened in all leads by increasing amounts of digitalis due to an early onset and acceleration of the repolarization process with a consequent reduction in duration of the T wave

C S T junction S-T segment and T wave In leads where the R wave constitutes the main phase of the QRS and the T wave is originally upright the earliest electrocardiographic changes are depression of the S-T segment reduction in voltage and duration of the T wave and slight shortening of the Q-T interval With cumulative dosage there is progressive shortening of the Q-T interval and depression of the S-T junction accompanied by downward bowing or sagging of the S-T segment and a change from an upright to a diphasic (- +) and finally an inverted T wave The reduction in Q-T interval is due in part to shortening of the S-T segment and in part to shortening of the terminal limb causing increased steepness of this portion of the deflection When digitalis retention comes within the range of full therapeutic to toxic doses the Q T interval is below normal limits the S T junction is significantly depressed the segment slopes obliquely downward in a straight line or in a slightly curved line with upward concavity to form the proximal limb of an inverted T wave, whose distal limb returns abruptly to the base line The foregoing changes are observed in left ventricular leads of normals and of patients with patterns of left ventricular hypertrophy and LBBB and in right ventricular leads in patients with patterns of right ventricular hypertrophy and RBBB When the original tracing before institution of therapy showed Q-T prolongation S-T depression and T wave inversion digitalization shortens the Q T to normal causes further S T depression and deeper inversion of the T wave and changes the contour of the S-T segment from a domelike convexity to a straight downslope or a concavely curved downslope With digitalizing or toxic doses leads with a predominant S wave display reciprocal elevation of the S T junction straightening of the S-T segment and rapid return of the distal limb of the upright T wave to the isoelectric line These changes are observed in right ventricular leads of normals and of patients with patterns of LVH and LBBB and sometimes in left ventricular leads in patients with RVH and RBBB Withdrawal of digitalis leads to gradual disappearance of these effects requiring from a few days to three to four weeks depending upon the rate of elimination of the preparation administered

D Other effects of digitalis

1 P wave — commonly shows no change but may be reduced in voltage

2 P R interval — is prolonged with slight overdosage Partial to complete atrioventricular block may result from gross overdosage

3 Rate and rhythm

a Normal sinus rhythm before digitalization

1) Therapeutic doses cause slight to moderate slowing

2) Toxic doses may cause

a) Ectopic beats Ventricular premature beats result from slight overdigitalization and tend to occur after each normal beat, producing a bigeminal (coupled) rhythm Larger doses may produce ectopic beats from multiple foci

b) Ectopic tachycardias Gross overdosage may produce auricular, nodal, or ventricular tachycardia, auricular or ventricular fibrillation Potassium depletion sensitizes the myocardium to the toxic effects of digitalis and predisposes to the development of the ectopic tachycardias, particularly auricular tachycardia with 2 1 ratio Bifocal ventricular tachycardia should always suggest gross overdigitalization

c) Sinoauricular or atrioventricular block

b Abnormal rhythms before digitalization

1) Ectopic beats associated with myocardial disease may disappear with therapeutic doses

2) Auricular flutter may be converted to auricular fibrillation

3) Auricular fibrillation Therapeutic doses produce moderate to marked slowing of the ventricular rate through depression of the atrioventricular node Toxic doses may produce a complete atrioventricular block with an idioventricular rhythm (page 113)

II QUINIDINE

A QRS complex is usually not altered by therapeutic doses but occasionally bundle branch block is produced

B Q-T interval is prolonged above the upper limits for normals principally by an increased duration of the T wave

C S-T junction – no significant change

D T wave is broadened reduced in amplitude and sometimes shallowly inverted

E Other effects Therapeutic doses may abolish auricular or ventricular tachycardia and auricular flutter or fibrillation while toxic doses may produce atrioventricular block bundle branch block ventricular premature beats or ventricular tachycardia

III CALCIUM The changes are related to the concentration of ionized calcium in the extracellular fluid

HYPOCALCEMIA	HYPERCALCEMIA
	QRS Complex
No significant change	No significant change in the usual clinical range of hypercalcemia Rapid intravenous administration of calcium may cause widening and slurring of the QRS
	Q-T Interval
Prolonged because of a marked increase in duration of the S-T segment	Abnormally short because of an absent or markedly shortened S-T segment

HYPOPCALCEMIA	HYPERCALCEMIA
S-T Junction and S-T Segment	
S T junction is not altered but the segment between the S T junction and T wave is characteristically straight horizontal and increased in duration reflecting an abnormal delay in the onset of repolarization	S-T segment is markedly shortened or absent due to an early onset of repolarization When the blood calcium is very high as in severe hyperparathyroidism repolarization may begin shortly before the completion of the QRS complex and consequently leads to a displacement of the S-T junction in the direction of the main deflection of the QRS
T Wave	
May become lower in amplitude or inverted	Little or nochange in clinical states with hypercalcemia Rapid intravenous administration of calcium may lead to flattening or disappearance of T waves
Other Effects	
	P waves — decreased in amplitude Bradycardia defective A-V conduction ventricular extrasystoles ventricular tachycardia and fibrillation may result from excessive intravenous administration of calcium

Restoration of normal blood calcium levels causes an immediate regression of these changes to normal

IV POTASSIUM

HYPOPOTASSEMIA	HYPERPOTASSEMIA
The electrocardiographic changes associated with clinical potassium deficiency are probably in part referable to a low intra cellular concentration of potassium which may or may not be *reflected in the serum* potassium level The word hypopotassemia here is taken to mean a state of potassium deficiency which produces electro cardiographic changes The abnormalities are aggravated by hypernatremia	The electrocardiographic changes associated with clinical potassium excess can be correlated fairly well with the extracellular concentration of potassium thereby permitting the use of the term hyperkaliemia The electrocardiographic alterations due to hyperpotassemia are enhanced by an associated low sodium level and are reduced by a normal or high plasma sodium concentration T waves resembling those of hyperkaliemia may be recorded as a manifestation of acute subepicardial injury with normal blood potassium presumably as a result of local extrusion of intracellular potassium
QRS Complex	
Usually no change Occasionally slight widening may be observed	Characteristic changes in the T wave precede alterations in QRS The first change in QRS occurs with levels in the vicinity of 8 mEq /L and consists in increase in amplitude and duration of the S wave in left precordial leads at the expense of the R With levels of 9-11 mEq /L intraventricular block of the RBBB type develops and with still higher levels

HYPOPOTASSEMIA	HYPERPOTASSEMIA
	defective intraventricular conduction progresses to the point of cardiac arrest Preterminally the QRS-T is fused into a smooth biphasic curve with marked slurring and widening of the QRS
S-T Junction and S-T Segment	
S-T Junction shows progressive depression in leads with a predominant R wave and often as well in leads with a predominant S wave The S-T segment sags downward from the depressed S-T junction in a U shaped curve to end in a low upright to inverted T	S-T junction may be depressed in leads with a predominant R wave The S-T segment retains its normal upward concavity, but becomes more steep in ascent as the amplitude *of the T wave increases When there is* defective intraventricular conduction, the S-T segment is directed obliquely upward in a straight line from the depressed S-T junction to the peak of the T wave
T Wave and U Wave	
With increasing hypopotassemia the T wave becomes lower in amplitude and finally inverted Concurrently the U wave becomes increasingly exaggerated as one or more humps to result in a double camel back or a *triple rolling contour The U wave tends to* fuse with the T wave, so that measurements of the Q-T interval are likely to be made to *the end of the U wave and to thus appear* erroneously prolonged Although fused in some leads the T and U waves are usually distinctive in other leads which are utilized for measurements	All normally upright T waves become characteristically church-steeple-like in contour *as plasma K rises above 6 5-7 0* mEq /L The T waves increase in amplitude and become sharper and more peaked as a *consequence the concave ascending and* descending limbs become steeper and the base appears narrower Although changes take *place in the T waves of all leads they are* not necessarily uniform The church steeple effect is usually greatest in leads from the *right precordium and transitional zone pro* vided that the T waves were originally upright in these leads Inverted T waves of *cavity origin (e g aV_R) become deeper and* conform to the reciprocal of the upright T waves Abnormally inverted T waves in leads *from the epicardium of either ventricle may* become less inverted and often upright In the absence of defective intraventricular conduction *the Q T interval is normal or slightly* decreased in duration With defective intraventricular conduction the Q-T interval is *prolonged*
Other Effects	
P-R interval may be slightly prolonged Ectopic auricular and/or *ventricular* rhythms may develop (premature systoles paroxysmal tachycardia flutter or fibrillation) Potassium depletion sensitizes the myocardium to the toxic effects of digitalis The	*P wave decreased in amplitude up to the point* of sinoauricular block and arrest of auricular activity at high potassium levels There is *a resultant shift to a ventricular pacemaker* this is at first regular but with increasing hyperkaliemia ventricular fibrillation may occur

HYPOPOTASSEMIA	HYPERPOTASSEMIA
combination of hypokaliemia and digitalization is prone to be complicated by ectopic tachycardias especially auricular tachycardia with 2 1 ratio and bifocal ventricular tachycardia	A-V node and ventricles Partial or complete atrioventricular block defective intraventricular conduction and ventricular standstill may occur at high levels Ventricular premature beats Hyperpotassemia will cause the disappearance of premature beats particularly when they result from digitalis intoxication

INDEX

This index merely covers major headings since each subdivision is readily accessible through the outline method of presentation and the cross references in the text

L

M

N

P

Q

V

W

V

W